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JUN 29 1948

THE AMERICAN JOURNAL of PSYCHIATRY

VOLUME 104
NUMBER 11
MAY 1948

Official Organ of
THE AMERICAN PSYCHIATRIC ASSOCIATION

Psychiatry

ITS EVOLUTION AND PRESENT STATUS

By William C. Menninger,
The Menninger Foundation

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—From the Preface

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124 ROBERTS PLACE, ITHACA, NEW YORK

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EFFECT OF THYROID THERAPY ON THE CONDITIONAL REFLEX FUNCTION IN HYPOTHYROIDISM[1]

W. HORSLEY GANTT, M.D., AND WALTER FLEISCHMANN, M.D.,
Baltimore, Md.

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The hypothyroid patient described below had the usual clinical and laboratory examinations before treatment and for 2 years after, and in addition special tests were made, viz., IQ, electroencephalic, conditional reflex. The correlation between these several factors in our patient reveals the quick dependence of some mental functions on thyroid therapy, and the parallel between the conditional reflex function and certain metabolic changes.

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> This boy developed normally until he was 6 years old. After that growth came almost to a standstill. Skin became rough and scaly and he became sluggish. At 10 years there was pain in the legs, gait became waddling. Brown spots appeared on skin at 10-11 years.
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> *Height:* 44.5 inches.
> *Height-age:* 5¾ years.
> *Skeletal proportions:* Immature for his age, corresponding to age of 5¾ years.
> *Osseous development:* Bone age of 5 years. No true epiphyseal dysgenesis but there is weakening and disintegration along the epiphyseal line of the heads of the femurs with separation and slipping of the femoral heads.
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> *Hair:* Sparse, dry, lusterless.
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> *Serum cholesterol:* 571 mg. %.
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> *Diuresis test for adrenal insufficiency:* Negative.

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The raised yellowish brown pigmented plaques on the face and the mole-like areas of pigmentation suggesting xanthomata are unusual; adrenal insufficiency was ruled out by the negative diuresis test.

The IQ (mental age of 11 years, IQ 81 Binet form L) showed marked scattering, a base line of 9 years with some tests at 10, at 11, at 12, at 13 and 14, and a vocabulary at 14. Those requiring immediate recall (digits, sentences, and word memories) were failed. The EEG was characterized by a slow pattern lacking the frequencies of 7 per second. High threshold for sensitivity, inability to carry out orders correctly, and a lack of the ability to form quickly new adaptive reactions were shown in the conditional reflex test.

Therapy was begun on 23 May 1944 with 32 mg. thyroid daily, increased after 2 June to 64 mg., on 16 June to 96 mg. and on 21 July to 128 mg. daily.

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lature is small and fairly firm. His color has improved somewhat although he still does not have very much pigmentation of the skin and the moles have not changed. The skin shows better circulation. There is considerable sweating and slight sudaminal rash over the chest. The hair has not changed. Teeth have not changed. Still walks with a marked waddling gait. No pain in hips. No apparent change in genitalia.

The patient, discharged on 27 July, was directed to continue 96 mg. thyroid daily.

Examination 14 April 1945: Patient continues to grow rapidly and has gained weight but is still 4.7 pounds below the average for his height. Active and energetic, brisk in his movements. Seems quite alert mentally. Posture and gait much improved, although there is still waddling.

He is of slender build with slender features and somewhat sunken eyes, average amount of subcutaneous fat. Tissues feel rather loose and the muscles are still rather small, somewhat flabby.

His lips and skin are of good color. The skin is everywhere warm, soft and very moist. The finger nails are very thin, almost paper-like, flat or almost concave.

There are now 7 permanent incisors and 2 upper bicuspids, two lower bicuspids are erupting, the 6-year molars are still present. The permanent teeth appear to be normally formed and enameled.

The genitalia are small and infantile. The right testis is in the scrotum, the left testis is in the lower part of the canal and the same size as the right; it could not be brought down into the scrotum.

Examination 9 February 1946: On thyroid 128 mg. daily, doing fine. Going to school every day, in the 9th grade. Has been well except that this morning after driving to Baltimore he vomited. He participates more actively in the life of the school, but whereas before therapy he was sluggish, now he gets into the usual skirmishes with pupils and teacher.

Measurements: Age 15 years, 8 months; height 50.2 inches; height-age 8¾ years; weight 22.7 kg. In good general condition; quiet and cooperative. Good color of lips and mucous membranes.

Dentition: In upper jaw are 7 permanent incisors, the canines, and 3 bicuspids; in the lower jaw, 1 canine and 1 bicuspid. All 6 year molars are present, but the 12-year molars have not erupted.

Sexual development: Right testis in scrotum, measures 2.2 x 1.2 cm. Left testis can be felt in the canal. Penis measures 6 x 2 cm. No pubic hair.

IQ 8 June 1946 was 80.

The Conditional Reflex Function

Against this background (a 13-year-old hypothyroid weighing 47 pounds, with B. M. R. changing from −36 to +11 and blood cholesterol from 571 mg. to 193 mg. within a month but with IQ remaining stable) there is a marked change in his ability to form conditional reflexes. A summary of these findings is given in Tables 1 and 2.

Method

The patient is first tested with the faradic current for threshold of perception in each hand and next for the threshold of pain. Then he is given 5 combinations of a certain colored light followed in 2″ by a shock and 5 combinations of another light without shock, there being an interval of 1 to 3 minutes between the lights. Evidence of spontaneous cr formation is noted in this period. Then he is instructed to press a bulb before the shock would come. If he gives no evidence of having formed the conditional reflex to the light, he is exposed to further repetitions of the combination of light and shock. The strength of the shock is regulated to the sensitivity of the individual—a crucial factor in this test distinguishing it from routine tests not related to the individual being tested.[2]

On subsequent tests the patient can be examined for the retention of what he has learned before, or by using new signals he may be examined for ability to form new conditional reflexes. Although it would seem that repetition makes it easier for the patient to form subsequent conditional reflexes, and that therefore the comparison cannot justly be made between the initial and subsequent tests, actually normal subjects show practically no improvement in forming conditional reflexes in this simple procedure after the first one is formed.

The conditional reflex function in this patient can be summarized as follows:

When first seen on 11 May 1944 he shows a very high threshold for both perception and pain to the faradic shock; thus the patient does not remove his hand from a strong shock of 100 volts and he gives scarcely any respiratory response. He has an almost complete inability to form any conditional reflexes and even shows little attempt to ex-

[2] The above is the simplest method of measuring the conditional reflex. No attempt is made here to present more than the bare outline of the method; this brief description does not take into account other observations and more complex measures (both muscular and visceral) for the detection of crs which appear in a more or less inhibited form.

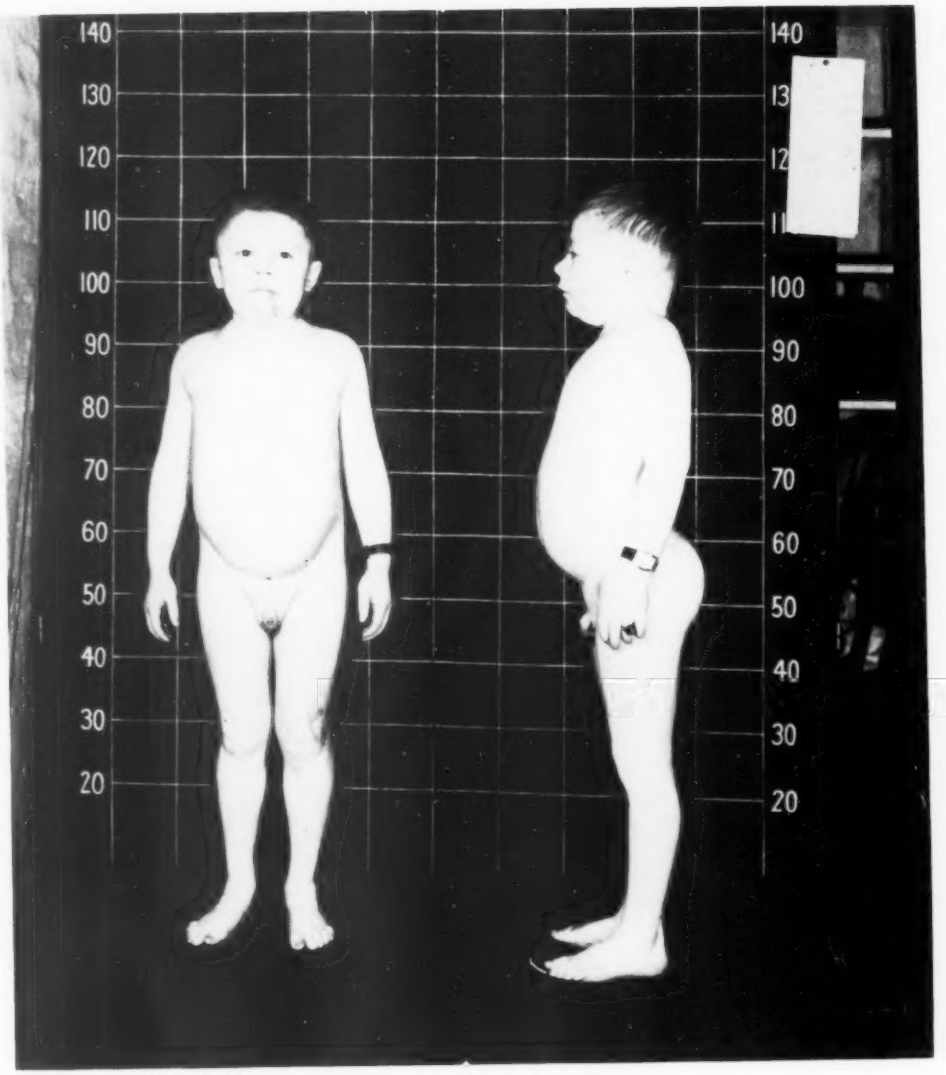

FIG. 1.—Patient P. R., May 1944, before thyroid therapy.

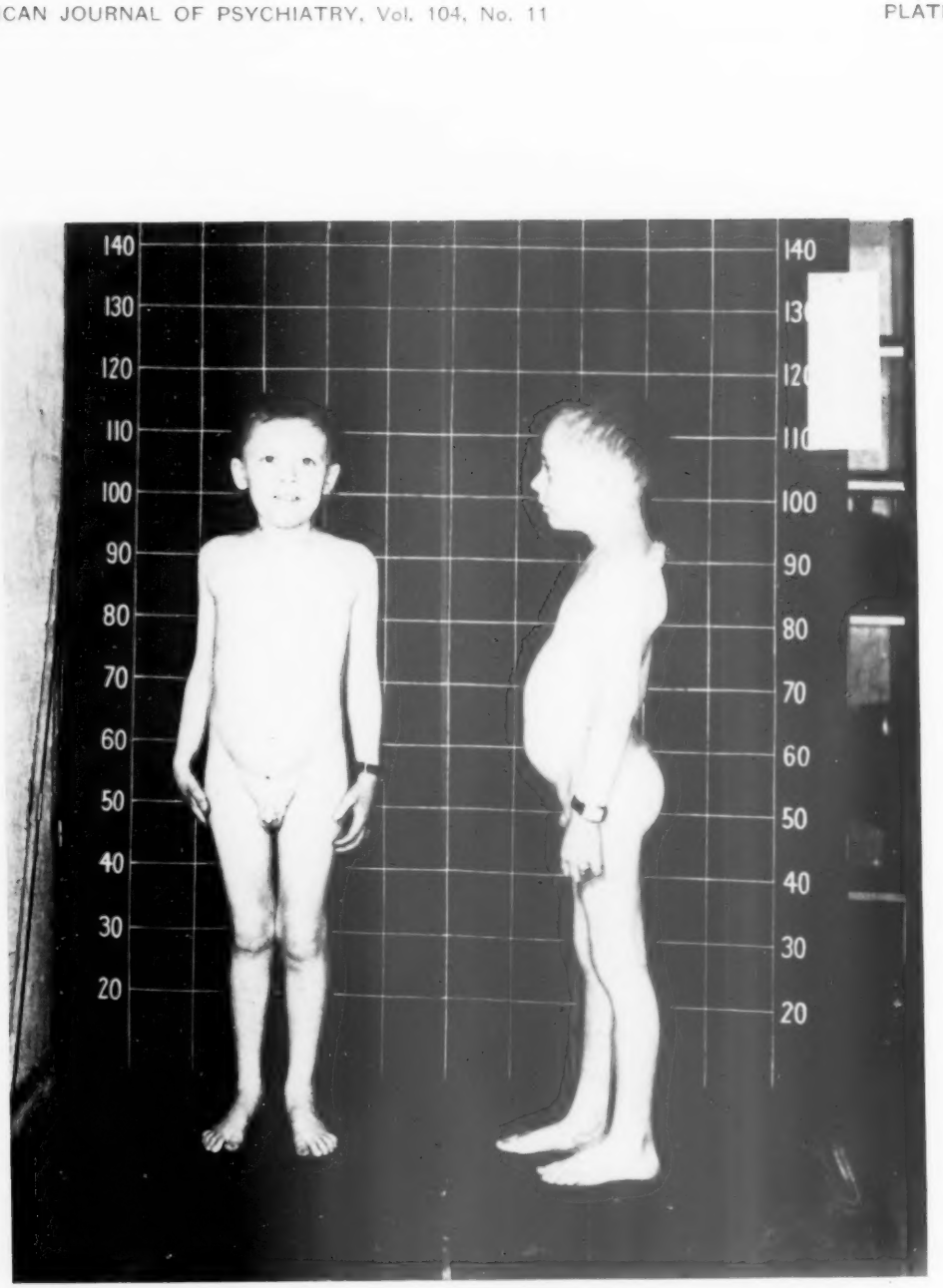

FIG. 2.—Patient P. R., 1946, 2 years after beginning thyroid therapy.

ecute orders, although he is cooperative. On 22 May the threshold to both perception and pain is much lower but still rather high for a young person. He is slightly more active but the excessive movements are still nonspecific and do not represent accurate conditional reflexes to the stimulus. There is rather high. But the patient has not only retained the conditional reflexes he formed a week ago but he shows the ability to form new conditional reflexes after only two repetitions. He still counts seconds as he did a week ago to aid in executing the movements at the proper time. On 21 July about 2

TABLE 1

	Age (yrs.-mos.)	Wt. kg.	Bone age	Height (height-age)	Mental age (IQ)	B. M. R.	Cholesterol	Treatment	EEG
5 May 1944..	13-11	24	5 yrs.	44.5 ($6\frac{1}{4}$)	11 yrs. (81)	−36	571	None.	Very slow pattern lacking frequ. 7/sec.
22 May	....	....	....	....	....	−36	522	None.	....
13 June	....	....	....	....	....	−9	243	Thyroid 32 mg. (start. May 23). Thyroid 64 mg. (start. June 2).	....
22 June	....	....	....	....	....	+11	193	Thyroid 96 mg. (start. 16 June).	Normal pattern frequ. 10/sec.
21 July	14-1	20.2	5 yrs.	45.4 ($6\frac{1}{2}$)	$11\frac{1}{3}$ yrs. (83)	+32	212	Thyroid 96 mg.	....
9 Feb. 1946..	15-8	22.2	8 yrs.	50.2 ($8\frac{3}{4}$)	....	....	185	Thyroid 128 mg. (start. July 21).	....
8 June	....	....	....	....	$12\frac{3}{4}$ yrs. (80)	....	...	....	....

TABLE 2

Date	Threshold: Sensitivity perception	Threshold: Pain	Cr formation	Differentiation	Latent period	Cr score	IQ
5 May 1944	57	115	None	None	....	20	81
22 May 1944	26	72	None	None	Uncertain	20	..
13 June 1944	22	87	+	+	3 inches	40	..
20 June 1944	17	75	+	+	....	80	..
21 July 1944	18	69	+	+	$\frac{1}{2}$ inch	90	83
9 Feb. 1946	8	39	+	+	$\frac{1}{2}$ inch	90	80

hardly any improvement in the specific conditional reflex from that seen on 11 May (Figs. 3 and 4).

On 13 June the patient's sensitivity is about the same as on 22 May but there is some improvement in his performance—he can form some conditional reflexes but executes them irregularly; his score improves for the first time. The respiration begins to show more irregularity than it had previously, indicating random activity.

On 20 June, a month after the beginning of therapy, a marked improvement has occurred. The threshold of sensitivity is still months after thyroid treatment began, the patient's performance with the conditional reflexes is about normal (Figs. 5, 6).

Eighteen months later, on 9 February 1946, the patient shows the same ability that he did in July 1944 to form conditional reflexes and to differentiate. The threshold for perception is about average but that to pain somewhat increased. Muscular activity is increased, tending toward abnormal hyperactivity which was not evident in 1944. The respiratory curve continues to be irregular (Fig. 7). The IQ remained almost exactly as it was before thyroid therapy (Fig. 8).

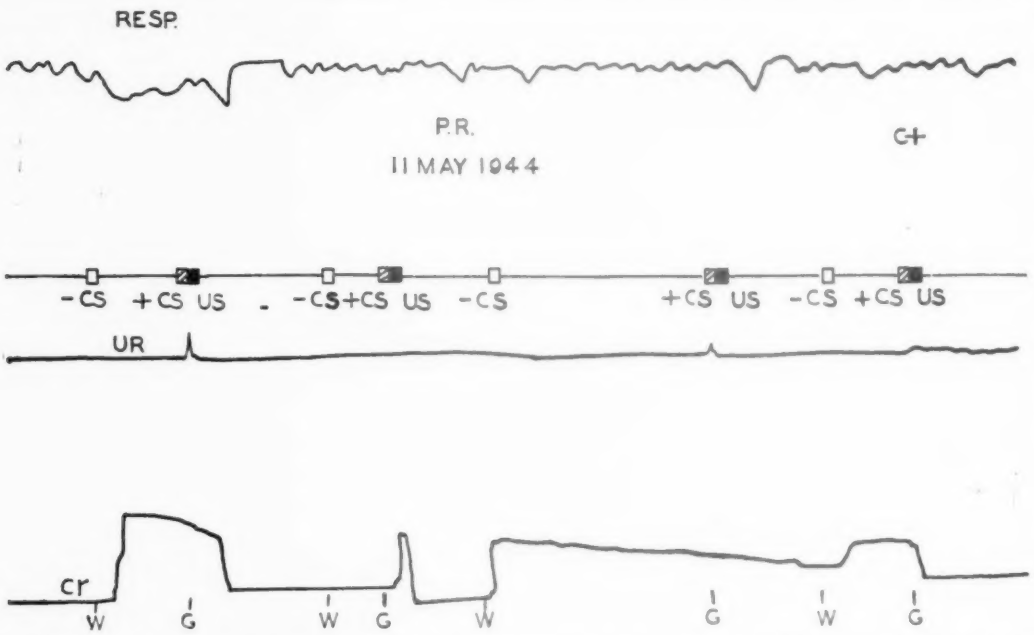

FIG. 3.—Cr record before therapy. Top line = respiration; +cs = positive conditional stimulus; −cs = negative conditional stimulus; US = unconditional stimulus (shock). Third line (UR) = movements of left hand on electrodes. Bottom line (cr) = movements of hand on bulb. W = white light (−cs); G = green light (+cs). Note diminished response to shock in UR, slow prolonged pressures of bulb (cr).

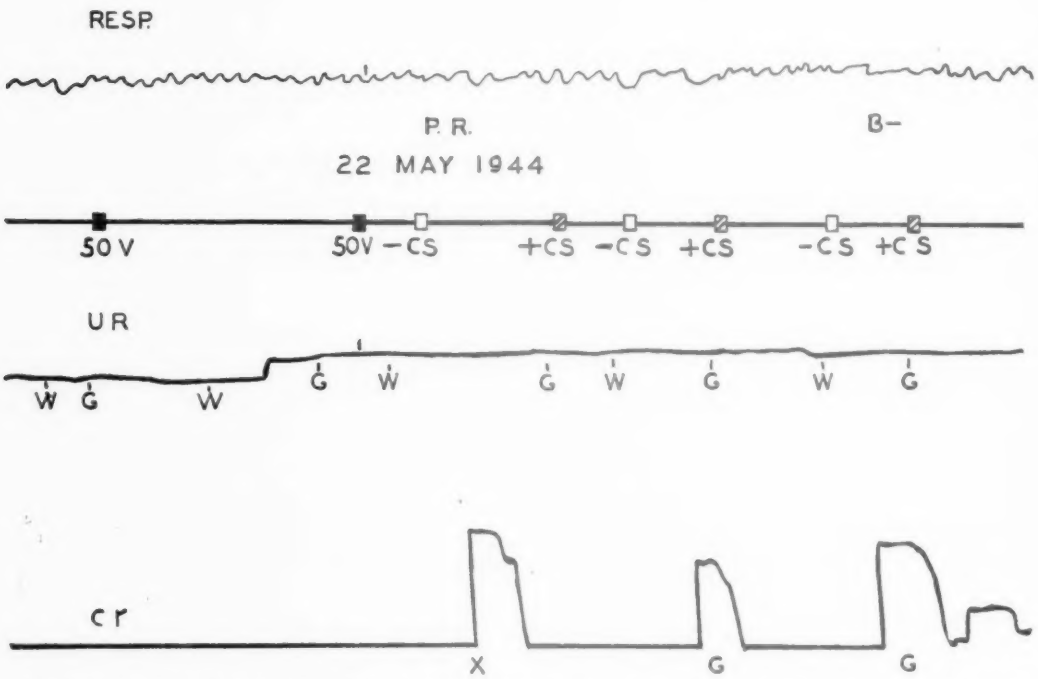

FIG. 4.—Cr record before therapy. Same symbols as Fig. 3. Note lack of response in both hands to the shock (50 volts) and same characteristics as in Fig. 3, also false pressure (x). The cr never occurs to the +cs but before.

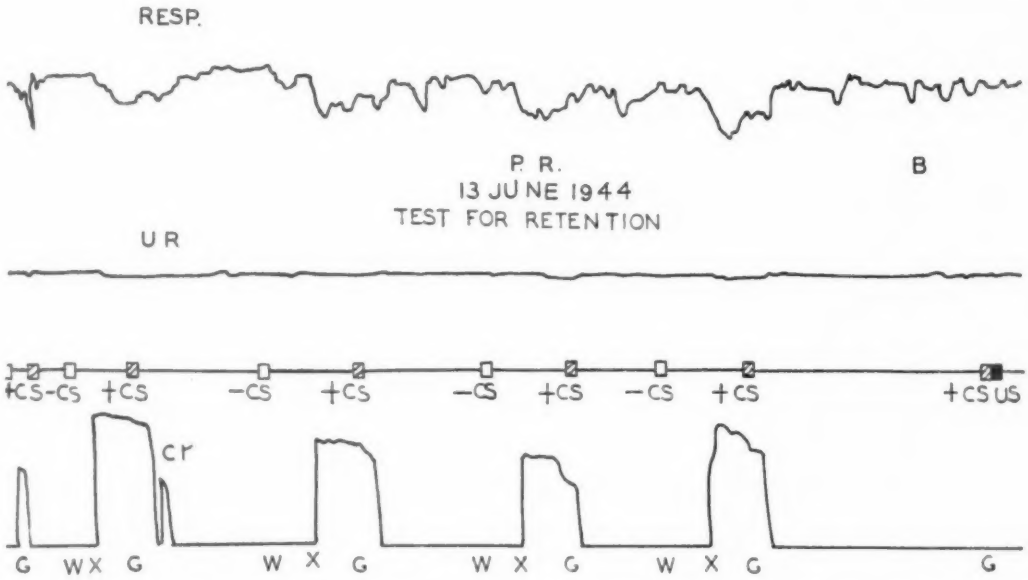

FIG. 5.—Three weeks after thyroid therapy. Note greater irregularity in respiration and presence of one or two correct crs, but mostly pressures during the intervals at x.

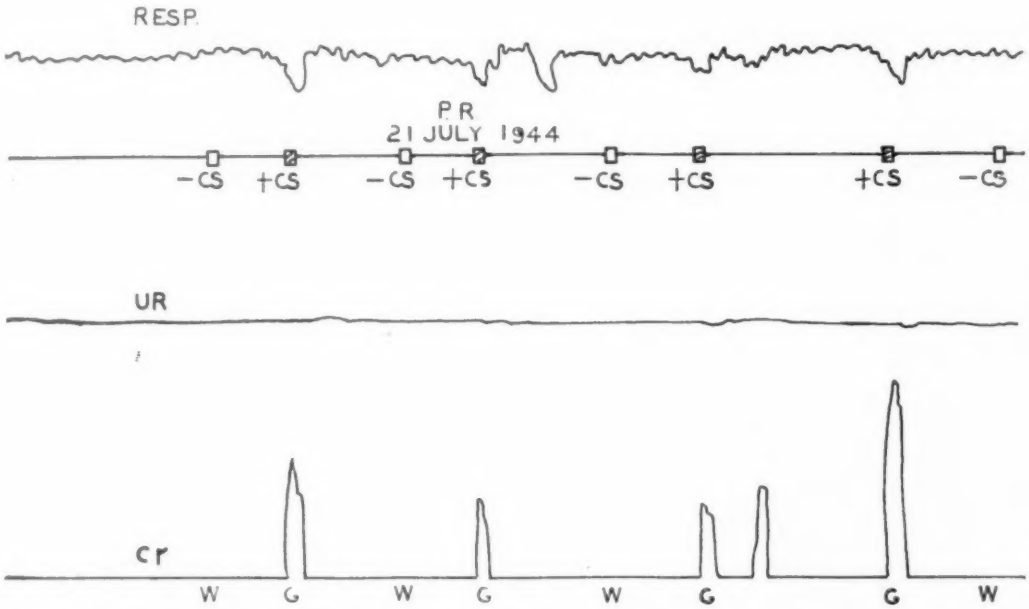

FIG. 6.—Two months after thyroid therapy. Notice definite crs to positive cs and good differentiation (no movement to W).

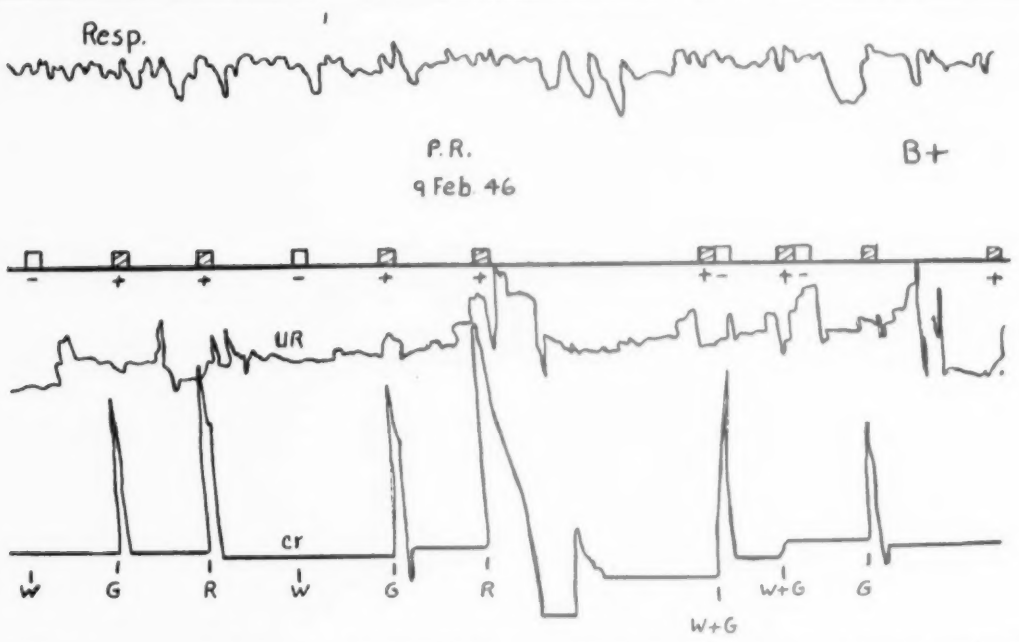

FIG. 7.—Cr record 18 months after thyroid therapy. Record is similar to Fig. 6 except respiration is more irregular, there is greater restlessness as indicated in movements 3rd line. Another +cs has been introduced (R). When the patient does not avoid the shock by the combination W + G on the 2d trial he tries not to press the bulb.

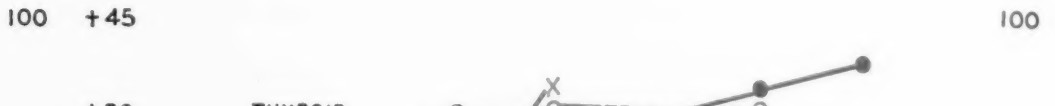

FIG. 8.—Fig. 8 shows the combination between the conditional reflex record, IQ, BMR, and blood cholesterol. For purposes of graphing, the conditional reflex scores are given a numerical score (A = 100, B = 80, C = 50, D = 25). The cholesterol is plotted on a scale opposite to the other items, viz., increasing downward, so that the *parallel* time changes are more easily apparent.

The increased sensitivity of the patient as treatment progresses can be seen not only in the conditional reflex function but even more markedly in the threshold for perception and pain to faradic stimulation (Fig. 9, Table 2).

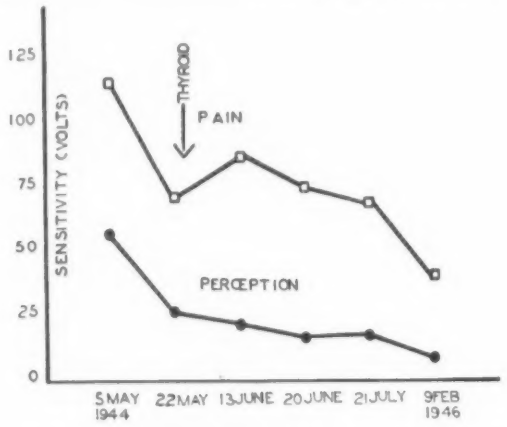

FIG. 9.—Change in threshold of sensitivity and threshold of pain in hypothyroid patient. Note the greatest drop is before thyroid therapy, probably due to the lowering of the threshold as a result of the patients having formed the conditional response to the shock.

DISCUSSION

That the threshold to pain and perception changes even before thyroid therapy indicates that this function depends upon many factors besides thyroid. Pain itself is a complex function composed of both an unconditional element and perhaps a more powerful conditional element—the result of the individual experience. In both human subjects and dogs it has been repeatedly observed by one of the authors (W. H. G.) that the threshold of pain diminishes markedly after the first experience owing to conditioning—the less intense stimuli which were not previously reacted to as painful become signals (conditional stimuli) of the stimulus which is to follow and they are reacted to as if they were painful. Owing to this conditional reflex it is doubtful whether we can see a true threshold for pain (with the usual methods) after the first trial. Subjectively speaking the fear of the pain has become equivalent to the pain itself.

The discrepancy between the constant threshold of pain seen by Harold Wolff and our decreasing threshold in normal subjects is probably due to the differences of methods employed—type of stimuli, opportunity for elaboration of conditional responses, etc. Dr. Wolff in commenting on these results states:

> Almost all human subjects will improve their ability to discriminate the just perceptible sensation of pain in successive trials. As in other quantitative measurements, a subject's first judgments are apt to be well over the threshold in order to be sure he is experiencing pain.
>
> When reaction to a noxious stimulus, such as wincing or withdrawal, or sweat, is chosen as an end point, instead of a verbal report of sensation, a wide range of end points is obtained. We made observations on ourselves (Hardy, Wolff, and Goodell) which indicate that reaction threshold to a stimulus which has been painful varies enormously from individual to individual and from day to day in the same individual. Reaction threshold is not the same as pain perception threshold. (Personal communication, 24 July 1947.)

Dr. Wolff's observation on the difference between perception and reaction is similar to what we have observed in the ability of patients in another connection; *i.e.*, perception and verbal reporting is a more stable function than the elaboration of new crs.

The extensive investigations concerned with the conditional reflex function have been carried out chiefly on animals; in only a few laboratories outside of Russia have patients been studied for conditional reflex function. But although the significance of the cr function in the various types of behavior deviations has not been generally recognized, researches have been made which indicate that this function bears a close relation to organic brain deficits, to toxic states, and to certain psychoses. Furthermore, this function has the advantage of being objectively observed and kymographically recorded.

Thus Gantt (2, 3) showed that the cr function was impaired in patients with brain damage (tumors, extirpations, etc.), or in alcoholic chronic states (Gantt and Muncie, 4) and that organic defects usually show more consistent impairment than do the so-called "functional" cases and that the patterns are different (2, 3, 4). However in not many patients has it been possible to see the results of therapy on quantitative metabolic functions and at the same time on the conditional reflex. The present case provides an unusual opportunity (in a patient with a known endocrine deficiency quantitatively modifiable through therapy) to correlate changes in

metabolism, as blood cholesterol and BMR, with the conditional reflex function.[3]

In comparing the conditional reflex function with (1) the BMR, blood cholesterol, and EEG and (2) the IQ, we see that there is a close parallel between ability to form conditional reflexes and the metabolic and electrical nervous changes, although the IQ remains unchanged (Fig. 8). This indicates that the ability to form conditional reflexes is a function closely dependent upon the *status præsens* of the organism, while the IQ is dependent upon the accumulation of learning over a number of years—apparently a much more stable function. A number of examinations of other patients bears out the greater stability of *retention* of conditional reflexes compared with present ability to form crs (3, 4). In fact all the tests involving *retention* of what was learned previously do not represent a complete test of present function: therefore they give us more of a picture of the patient's history than of what he can do at the time of observation. The various mental tests do sometimes reveal in their different parts now present ability, now past accumulation of learning, but the unanalyzed aggregate score is usually worthless for evaluating the patient as he faces us. It is as if one took photographs of a patient every 5 years and then compounded them into one. With the conditional reflex test, some elements such as retention depend at least in part upon past ability, but there is a clear distinction between (1) the ability to form conditional reflexes and to differentiate them during the period of examination and (2) the retention of previously formed conditional reflexes.[4]

The argument can be advanced that the *mental age* of the patient increased in the 2 years after thyroid therapy though his IQ remained the same. Owing to the complexity of the problem involving mental age and IQ in a growing person, this question cannot be settled here, but it is nevertheless significant that neither mental age nor IQ changed between 5 May and 21 July 1944 during the period of rapid change in blood cholesterol, BMR, conditional reflex function, and sensitivity (Figs. 8, 9).

The school record of this patient provides perhaps a commentary on criteria in our educational system rather than anything else. For here was a marked cretin, too sluggish to form simple adaptive reactions, who nevertheless passed all his grades.

Several previous studies on conditional reflexes in both hypothyroid animals and patients parallel ours. Liddell, investigating the conditional reflex impairment in thyroidectomized sheep, found a great increase in reaction time, a much longer period to solve labyrinth problems in the cretin (313 trials compared with the normal of 156), and although no change in formation or extinction of the conditional reflexes, there was "weakening of the inhibitory processes in the cortex." Asimov(1), in thyroidectomized dogs, showed that the already formed conditional reflexes were weakened, improved only slightly with practice, and were unstable. Shastin(10), in the laboratory of Krasnogorsky, found in myxedematous children that the salivary conditional reflex was elaborated only after 106 combinations with the unconditional stimulus compared to 15-20 in the normal child and that the latent period was twice as long as the normal.

Himwich et al.(7) found in 11 cretins that after thyroid therapy there was an accelerated cerebral blood flow (57%) and an increase of 32% in cerebral metabolism with altered cerebral electrical potentials. "Psychological improvement was either absent or of questionable significance." He noted slight increase in speed of tapping. The failure to find improvement in learning was probably

[3] Halstead, Rappaport, and others have introduced tests for the study of organic brain damage which, though not performed on the pattern of the cr test, bring additional clearcut and objective evidence for the dependence of higher nervous function on unimpaired brain tissue. The general lack of such tests has resulted in much confusion in evaluation of true brain damage and in differentiating between the organic or so-called functional states (6).

[4] Leslie Hohman suggests that the change of conditional reflex function against the background of a constant IQ results from the fact that the conditional reflex represents a more primitive function than does the IQ. This point of view deserves consideration; but in my studies(2, 3) the ability to form and differentiate conditional reflexes is a *more delicate* function than that of retention of previously formed responses, upon which much of the Binet-Simon depends.

a result of the kind of psychological test used, which was not stated further than "a battery of tests." His results are in close agreement with ours except that our cr record was parallel to the change that he found in cerebral metabolism and not to his psychological tests; the IQ and school record were representative of the latter.

As further evidence that the conditional reflex function is a delicate measure of the effects of metabolic and other changes affecting the higher nervous activity, are the results of Wintrobe and Gantt(5) in the study of vitamin B deficiencies. After withholding some elements of the vitamin B complex, there was an impairment of the ability to form conditional reflexes as soon as 4 days after the deprivation, although no other change in nervous activity could be detected until weeks or months later.

Furthermore, the results of such a delicate test for present function indicate that most of the improvement in conditional reflex function comes within a short period—days or weeks—after the beginning of thyroid ingestion. After 2 years no further improvement of the reactions tested had occurred. With a more nearly normal learning ability, however, it is to be expected that, though the speed of learning does not increase, there will be an accumulation over the years which eventually might be reflected in a test such as the Binet-Simon.

To claim that the described cr function is the only delicate index of mental function is not our purpose but rather to emphasize that it represents a form of higher nervous activity which parallels BMR and cholesterol changes and hence can be used to detect and study nervous functions which are not usually assessed.

Summary

A complete clinical study of a hypothyroid boy, age 13, was made for a period of 2 years. An examination of the higher nervous activity by the conditional reflex method showed that the ability to form and differentiate conditional reflexes ran parallel to the metabolic records (BMR, serum cholesterol) and to the EEG, showed a marked improvement within several weeks after thyroid therapy, the improvement in these items reaching a level in 2 months, without further change after 2 years of therapy. The IQ (depending largely upon an accumulation of learning) showed a marked stability over the same 2-year period. The conditional reflex, on the contrary, is a delicate measure of higher nervous function practically independent of education, related specifically to the *status praesens* of the patient and in this hypothyroid patient to basic metabolic changes.

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COMBINED INSULIN COMA AND ELECTRIC CONVULSIVE THERAPY IN SCHIZOPHRENIA [1]

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The use of combined insulin-electric shock in schizophrenia has become more widespread, but reports as to its efficiency are meagre. It is now generally accepted that both insulin and convulsive therapy used separately are of value in the treatment of schizophrenia though failures are considerable and relapses frequent. Investigators looking for a means of obtaining longer-lasting remissions with greater frequency then attempted combining these therapies.

Georgi(1) in 1937 introduced metrazol convulsive therapy during insulin hypoglycemia because of theoretical considerations and because of the observation that following "spontaneous" convulsions during hypoglycemia there was a sudden marked improvement in the patient's mental condition. Metrazol was replaced by the electric induced convulsion because of its advantages and is given during the hypoglycemic state at the time the insulin coma would have been terminated, *i. e.*, usually during the fourth hour of treatment.

The fear that the respiratory difficulties frequent in hypoglycemia might be aggravated by the depressant respiratory effect of the electrically induced seizure was not substantiated, and no particular difficulties were encountered. The restraining sheet under which our insulin patients are kept was loosened in order to facilitate artificial respiration or intravenous injections if necessary. Caution was taken that the extremities were not in an awkward position which might favor muscle or bone complications. Frequently it was impossible to insert the mouth gag; this was therefore inserted quickly during the tonic phase if possible. The hypoglycemia was terminated routinely shortly after the convulsion by glucose. This was by nasal tube inserted prior to the electric shock treatment and its correct position in the stomach verified once more by the withdrawal of gastric juice after the convulsion.

The amount of electrical energy necessary to produce a seizure varies with the depth of the hypoglycemic state. While hypoglycemia lowers the convulsive threshold, a deep coma increases this threshold considerably. This was demonstrated by inducing convulsions in different stages of coma. In the very deep states of hypoglycemia, characterized by loss of corneal reflexes and miotic pupils, several times it was impossible to induce any convulsion: a situation which was never encountered in our experience with ordinary electric shock therapy. The resulting convulsions are more often atypical and modified by the comatose state. The most frequent modification is the absence of the clonic phase. The patient has only a tonic contraction followed by few if any clonic movements. This accords with the theory that the clonic phase characterizes the regaining of functioning of the cerebral cortex, which is considered deeply deranged in hypoglycemic coma. Another less frequent modification observed is the absence of the tonic phase with a few clonic movements occurring immediately after the stimulus. It was surprising that the respiratory pattern of the convulsion was hardly influenced by the hypoglycemic coma. The usual deep, stertorous breathing at the end of the convulsion appeared even in patients who had very shallow respirations during the preceding hypoglycemic coma. There were instances of respiratory difficulties but they were hardly more frequent than in ordinary electric shock therapy. No serious cardiovascular episodes were observed in our group. No fatalities occurred. There were 2 mild prolonged comas and 2 patients suffered mild thoracic compression fractures.

Each patient was given 5 or 6 insulin treatments weekly, irrespective of how many times a week electric shock was added to it. Combined treatments were first given twice a week, more recently 3 times weekly, and in

[1] Read at the 103d annual meeting of The American Psychiatric Association, New York, N. Y., May 19-23, 1947.

2 very disturbed patients daily for 4 or 5 treatments. The combined treatment was usually started toward the latter half of the course of the insulin coma treatment, which usually consisted of 50 comas. The total number of combined treatments varied from 6 to 20 and averaged about 10 per patient.

The majority of our earlier patients were chronic or subchronic schizophrenic cases that had responded unfavorably to a full course of 20 electric shock treatments or 40-50 insulin comas or both successively. However, as our experience increased the selection of cases shifted to more favorable schizophrenics.

Our total group consists of 80 patients varying in age from 13-48 treated with combined shock over a period between July, 1942 until January, 1947. Of these, 78 were schizophrenics, 2 nonschizophrenic. Of the 78 schizophrenics more than one-half (42) had received some form of shock therapy previously, and almost all of this group of 42 had more than one kind of therapy. This included electric shock, insulin coma, ambulatory insulin plus electric shock, etc.

In evaluating the results for the total group one must take into consideration the highly unfavorable cases first selected—cases with poor prognosis and poor results from previous treatments. The results of the treatment were compared for 3 time periods: (1) immediately after completion of treatment, (2) 2-3 months after treatment was terminated, and (3) the latest available follow-up.

1. The results of the group immediately following treatment are as follows: 78 schizophrenics, 1 recovered, 21 much improved, 28 improved, and 28 unimproved.

2. Reviewing the group at a period 2-3 months later, the trend was towards a less successful result: recovered, 1; much improved, 17 instead of 21; improved, 20 instead of 28; unimproved, 40 as against original 28. For this group of 78 schizophrenics this represents a rate of considerable improvement of only 28%. When this group is broken down into individual cases evaluated according to duration of illness, this poor proportion of successful responses is seen as not measuring the efficacy of the treatment in general. Of the 13 patients who were ill less than 6 months, the response in 10 was satisfactory—or a favorable rate of 77%. Of the 28 patients whose duration of illness was 1 year or less (8 of whom had previous shock treatment), 54% (15 cases) gave a satisfactory response. Of the 40 patients whose duration of illness was 2 years or less (15 of whom had previous treatment) 16 or 40% gave a satisfactory response. Our results then approximate the level of success of insulin coma treatment as well as some reported results in convulsive therapy but are not as favorable as the results previously reported by Chapuis(2), and Chapuis and Georgi(3) for combined insulin-convulsive treatment. This may be explained by the different material, because our hospital admits only voluntary patients, and this makes for a selected group excluding more acutely psychotic patients. It is generally known that patients with acute onset and active and dramatic symptomatology respond better to shock therapy than those with an insidious course and less dramatic symptomatology.

In evaluating the duration of illness together with the factor of previous shock treatment, of those with the *same* duration of illness the more favorable results occurred in patients not previously treated by shock. This difference, however, was not noted in patients sick less than 6 months, both groups showing similar favorable results. As we approach the group showing longer duration of illness but still under 1 year, the finding of previous shock treatment was more common in the patients responding less favorably. Of these 28 patients, 55% of those without previous treatment responded well while only 37.5% of those with previous treatment showed favorable results. In the next group, those sick 2 years or less, the previously untreated group (now 24 patients) has a favorable response of 46%, while the previously treated group (16 patients) has a favorable response of 25%. However, in this group, which has a second year of illness, there were few good responses in general.

Thus in our total group the best responses occurred in patients sick less than 1 year who previously had no shock treatment. A pertinent question is whether these cases who had had previous shock treatment failed to respond favorably to combined treatment because of actual changes created by previ-

ous shock or because they constitute that well-known group of refractive cases unresponsive to any treatment.

Continuing evaluation of duration of illness, the patients who had been sick 2 years or more (38) responded unfavorably. Of these 38, 3 (8%) were much improved, at the present time, the interval since treatment varying from 6 months to 3½ years. Because of this some patients show an improvement of 2-3 years while other patients, though maintaining their improvement, are only 6 months away from treatment. Of the total series, 22 could now be considered much

RESULTS CORRELATED WITH DURATION OF ILLNESS AND PREVIOUS SHOCK THERAPY

	Previously untreated group		Previously treated group		Total	
SICK 6 MONTHS						
Recovered	1	= 78%	0	= 75%	1	= 77%
Much improved	6		3		9	
Improved	0		0		0	
Unimproved	2		1		3	
Total	9		4		13	
SICK LESS THAN 1 YEAR						
Recovered	1	= 60%	0	= 37.5%	1	= 54%
Much improved	11		3		14	
Improved	1		1		2	
Unimproved	7		4		11	
Total	20		8		28	
SICK LESS THAN 2 YEARS						
Recovered	1	= 50%	0	= 25%	1	= 40%
Much improved	11		4		15	
Improved	3		5		8	
Unimproved	9		7		16	
Total	24		16		40	
SICK TWO YEARS OR MORE						
Recovered	0	= 9%	0	= 7%	0	= 8%
Much improved	1		2		3	
Improved	5		6		11	
Unimproved	5		19		24	
Total	11		27		38	

which lasted only a few months; 11 showed mild amelioration or improvement sustained in half; and 24 were entirely unimproved.

Of the entire series of 78 schizophrenics, 28 were committed to various state hospitals, the majority within one year after discharge, and most of them (19) came from that unfavorable group who had been ill for over 2 years and who had an over-all picture of much previous treatment. Most of these patients are still in hospitals; a few (3) have been paroled and are adjusting in a limited manner.

3. The entire series was again rechecked improved or recovered, 14 improved, and 42 unimproved.

In breaking down these figures once more in relation to the duration of illness, we again evaluate the 18 patients who were discharged in a state of social remission, either much improved or recovered: 15 who came from the group sick less than 2 years, 3 from the group sick over 2 years. Of the 15 cases with shorter duration of illness, who had done so well, 12 of them maintained their status quo, some for several years. But the 3 previously in a state of social remission who had been ill 2 years or more prior to

treatment lost their state of improvement within a few months.

Thus again we see a greater tendency to stabilization in the least chronic group. The group of 78 schizophrenics included all types: about one-half were hebephrenic, one quarter catatonics, the rest distributed over the other subtypes. There are too few cases that responded well to the treatment in general to designate convincingly the most favorable type, but the impression was that, proportionately, catatonics responded best.

In summary we might say that combined insulin-electric shock is a safe method of treatment with hardly any more difficulties than insulin shock therapy alone. It is still doubtful whether it is a more effective therapy than insulin shock alone or intensive convulsive therapy even though some patients refractive to insulin alone responded well to combined treatment. A satisfactory therapeutic result is best achieved in patients in whom the treatment is begun within the first 6 months. Our results for this group corroborate those given by Chapuis and Georgi(3) in obtaining remissions in about three-fourths of the patients treated. When the duration of illness is up to one year we still obtain a good percentage of satisfactory responses, especially in patients who had not been exposed to other shock therapies previously. Beyond one year there is a decrease of effectiveness.

Beyond 2 years, satisfactory therapeutic results have not occurred, in that, if a considerable degree of improvement is obtained, it is not lasting; more often there is either no improvement or only a mild amelioration of symptoms, which again is only temporary. In this chronic group our results do not corroborate the striking results of Chapuis and Georgi who report social and even total remissions in 25-30% of those patients whose illness was of more than 2 years duration; a large percentage of these they claim are maintained for several years.

The stability of our remissions can hardly be judged from our shorter period of experience but seems related inversely to the duration of the illness prior to treatment. Relapses are less common in patients with the shortest duration of illness and occurred in all of the few patients with more than 2 years where a remission had been achieved.

The question can be raised whether combined insulin-electric shock therapy is not the treatment of choice in all schizophrenics. This may well be; but practical considerations will make it impossible to apply this procedure to all schizophrenics from the beginning of their illness. Many can be brought to a lasting remission with the simpler electric shock therapy. Those preferring insulin therapy have usually been applying combined treatment and their results are among the best reported so far. As a whole, it appears that intense treatment is the most important requirement in schizophrenia. The choice of one of the shock methods for an individual case remains arbitrary, but the possibility of combining insulin with the simple electric-convulsive technique is a useful way to give difficult cases the benefit of the most intensive treatment.

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PSYCHOPHARMACOLOGIC STUDY OF SCHIZOPHRENIA AND DEPRESSION

IV. The Influence of Electric Convulsive Therapy on the Sodium Amytal Response of the Electroencephalogram [1]

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Traditionally, the investigation of electroencephalographic correlates of psychiatric conditions has dealt almost exclusively with measurements of either alpha (8 to 13 per sec.) or delta (less than 6 per sec.) activity. Recently, however, several investigators have turned their attention to the faster, beta bands (above 13 or 14 per sec.).

Hoagland and associates(1) have recently reported that they observed a high degree of relationship between the diminution of beta activity in the electroencephalograms of patients with involutional psychoses and improvement in clinical status as the patients underwent a course of electric convulsive therapy.

Cohn and Katzenelbogen(2), Rubin, Malamud and Hope(3), and Brazier and Finesinger(4) have studied the changes induced in the EEG after administration of intravenous sodium amytal. These changes were manifest in a pronounced increase in beta activity.

Sodium amytal is known to have a temporary beneficial effect upon the symptoms of some psychotic patients. These transient effects have been shown to be most marked in patients with pathologic depression and schizophrenia, although there appears to be a differential effect in these two disorders. Patients with schizophrenia are characterized by the rapid development of "psychologic" tolerance to the drug, in terms of type and duration of their psychologic response. In contrast, those with pathologic depression do not develop such a tolerance(5).

These various observations seem to lead us into a dilemma, for, on the one hand, it appears that, associated with electric convulsive therapy, a decrease in psychotic symptoms is accompanied by a decrease in the amount of fast activity in the EEG, while, on the other hand, it appears that a drug (sodium amytal) which induces transient improvement in the clinical state also induces an increase in fast activity in the EEG.

The data to be presented here have some bearing on these incompatible observations. Furthermore, they provide evidence pertaining to tolerance and to the effects of electric convulsive therapy. Specifically, this study deals with the concomitant changes in the beta activity of the EEG and psychologic changes induced by repeated intravenous administration of sodium amytal (plus racemic amphetamine sulfate) and concomitant changes after completion of a course of electric convulsive therapy.

Method

Nine patients suitable for testing (5 with schizophrenia and 4 with depression) were subjected to the routine procedure. Suitability for testing depended upon each patient meeting four requirements: (1) The patient must present typical signs and symptoms of one or the other disorder; (2) he must not have been taking barbiturates for some time before admission; (3) he must not have previously been subjected to any of the shock therapies (insulin, metrazol, or electric); and (4) he must be cooperative in the experimental situation.

The experimental procedure was as follows: Each patient was taken to the electroencephalographic laboratory one hour after breakfast. Cortical potentials were secured with standard equipment. Monopolar leads from the left occipital, motor, and frontal areas were used for analysis. After a one-

[1] Read at the 103d annual meeting of The American Psychiatric Association, New York, N. Y., May 19-23, 1947.

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half hour control recording, 250 mg. of sodium amytal was injected intravenously during 3 minutes, followed by 20 mg. of racemic amphetamine sulfate. The type of psychologic response was noted and the electroencephalographic recording was continued for one hour.

The above procedure was continued for 5 consecutive days.

The patients were then subjected to electric convulsive therapy. In accordance with hospital routine, patients having schizophrenia received 3 treatments per week (9, 15, 15, 15, and 15 respectively), while those with depression received 2 per week (8, 5, 8, and 6 respectively). (Unfortunately, the number of convulsions induced in each patient was dependent upon the clinical response and not the formulation of this study.) Approximately one week following the last convulsion, the patients were again subjected to the routine electroencephalographic recording in response to the drugs for a single observational period.

The electric convulsive therapy was administered by the usual technique, using 60-cycle alternating current, voltage held constant at 120 volts, the time varying with that needed for the individual patient.

Reactions to the drugs were evaluated in those with schizophrenia as good, moderately good, and poor, in accordance with the degree of improvement in affect, associations, and insight. For patients with depression, the reactions were defined in accordance with the degree of shift of the affective state, retardation, or agitation, whichever was present. A complete description of this evaluational technique has been given elsewhere(5).

The electroencephalographic records were analyzed as follows: Before the drugs were injected, and at intervals of 5, 15, 25, 35, and 45 minutes thereafter, the amount of beta activity in the occipital, motor, and frontal areas was computed. Beta activity was arbitrarily defined here as falling above 15 per sec. in frequency, having an amplitude above 5 microvolts, and occurring in organized rhythmic bursts of 3 or more waves. Three samples, each 1 meter long (representing a run of 33.3 seconds) were selected at random for analysis from the pre-injection record. In the post-injection records, the samples were taken at the 5, 15, 25, 35, and 45 minute points. Separate analyses of the 3 cortical areas (frontal, motor, and occipital) were made. The number of centimeters in each sample occupied by beta activity was determined, providing, directly, percent time beta activity; the average of the 3 samples was used as the beta activity index in the final computation of the data.

Results

The quantitative results are presented in Tables 1, 2, 3, 4, and 5 and in Figs. 1, 2, and 3.

It is quite apparent (Fig. 1) that the administration of sodium amytal has the immediate, and fairly long-lasting, effect of increasing the per cent time beta in the EEG (Tables 1, 2, and 3), the peak appearing at the 5-minute interval, with a gradually subsiding curve up to and beyond 45 minutes. This was true for all patients and all areas recorded, except for 2 cases with schizophrenia in whom the occipital per cent time peak was delayed, appearing at the 15- rather than the 5-minute interval (Table 1). These observations are in accord with those of other investigators.

The data also indicate the presence of an areal gradient (Fig. 2), frontal-motor-occipital, in the susceptibility to increased per cent time beta after administration of the selected drugs (Tables 1, 2, and 3). This gradient was discussed by Brazier and Finesinger(4). As seen 5 minutes after the injection of sodium amytal, the areal gradient, in the case of the depressed group, appears as an accentuation of the control period gradient. However, in the schizophrenic patients the areal gradient appears to be created by the drug, as their control period shows a relatively flat frontal-motor-occipital per cent time curve. Two patients with depression exhibited a partial exception to this generalization, in that their motor and occipital values were identical, although the frontal values were higher (Table 2).

These findings persist in spite of the great extent of individual differences present in the per cent time beta values.

When the minute-to-minute values of per cent time beta are inspected on each of the experimental days for both groups, it may be

TABLE 1

THE MEAN PER CENT BETA ACTIVITY FOR EACH PATIENT WITH SCHIZOPHRENIA BEFORE AND AT VARIOUS TIMES AFTER INJECTION OF SODIUM AMYTAL AND RACEMIC AMPHETAMINE SULFATE FOR FIVE CONSECUTIVE DAYS COMPARED TO THE BETA ACTIVITY RESPONSE AFTER ELECTRIC CONVULSIVE THERAPY

		Before electric convulsive therapy								After electric convulsive therapy					
			Minutes after injection								Minutes after injection				
Case	Area	Pre-injection	5	15	25	35	45	No. of convulsions	No. of days after last convulsion	Pre-injection	5	15	25	35	45
Bu	Frontal	10.36	59.68	49.68	34.86	24.98	19.68	9	5	9.2	16.0	13.0	8.1	9.3	8.3
	Motor	4.16	47.92	38.38	29.40	20.14	18.70			2.0	12.2	9.5	8.9	8.7	6.7
	Occipital	3.70	39.50	29.08	20.92	12.08	9.90			2.9	6.0	7.2	4.5	4.9	3.7
Jo	Frontal	10.80	44.78	31.66	29.20	29.38	24.88	15	7	4.7	13.6	11.0	9.4	8.9	8.4
	Motor	4.50	34.98	21.04	18.16	17.38	15.80			6.4	23.3	15.2	13.3	15.1	11.5
	Occipital	9.14	23.66	15.76	15.20	13.52	13.60			11.0	10.5	9.0	9.1	10.3	9.4
Pe	Frontal	20.06	40.70	39.10	33.12	33.18	31.46	15	5	3.5	9.0	11.6	12.3	13.5	14.3
	Motor	20.18	33.38	36.38	31.72	31.68	30.28			5.8	12.5	13.4	15.1	13.6	15.1
	Occipital	24.48	24.40	28.96	26.00	26.16	27.56			5.3	8.7	5.4	8.2	11.0	7.8
Ne	Frontal	2.40	36.60	12.74	6.92	6.24	6.24	15	10	4.2	4.7	4.9	4.3	4.2	4.2
	Motor	2.20	15.30	5.82	3.64	3.38	3.38			1.6	3.7	4.7	4.1	4.0	4.0
	Occipital	2.84	11.58	4.50	2.72	2.40	2.40			1.7	1.4	2.5	1.8	2.6	2.6
Ba	Frontal	9.18	23.96	21.42	19.84	15.16	12.72	15	8	1.4	4.9	3.8	2.0	2.3	1.4
	Motor	6.64	13.06	13.82	13.10	11.06	10.10			0.5	1.3	0.8	0.7	0.5	0.6
	Occipital	8.68	9.12	10.62	10.84	9.30	9.58			0.4	0.4	0.2	0.2	0.2	0.2

TABLE 2

THE MEAN PER CENT BETA ACTIVITY FOR EACH PATIENT WITH DEPRESSION BEFORE AND AT VARIOUS TIMES AFTER INJECTION OF SODIUM AMYTAL AND RACEMIC AMPHETAMINE SULFATE FOR FIVE CONSECUTIVE DAYS COMPARED TO THE BETA ACTIVITY RESPONSE AFTER ELECTRIC CONVULSIVE THERAPY

		Before electric convulsive therapy								After electric convulsive therapy					
			Minutes after injection								Minutes after injection				
Case	Area	Pre-injection	5	15	25	35	45	No. of convulsions	No. of days after last convulsion	Pre-injection	5	15	25	35	45
Pe	Frontal	13.94	52.84	39.32	34.98	32.78	24.16	8	6	5.2	20.5	17.9	11.8	10.2	8.6
	Motor	8.42	43.60	36.06	31.94	31.60	26.86			10.7	29.4	26.7	24.8	21.6	14.7
	Occipital	4.06	22.42	13.70	9.64	9.46	7.46			1.7	2.6	3.1	2.3	2.1	2.3
Mz	Frontal	11.14	38.06	35.36	30.84	27.06	25.94	5	9	6.3	19.2	18.3	16.4	13.2	10.6
	Motor	6.16	33.64	25.82	23.74	22.50	21.28			6.6	22.5	19.3	20.2	20.4	17.7
	Occipital	3.12	8.58	6.98	6.08	5.38	3.78			1.5	3.4	1.8	1.1	1.6	0.9
Bl	Frontal	5.68	27.64	14.80	11.98	13.32	10.52	8	5	2.6	8.8	4.0	5.3	3.8	2.8
	Motor	2.58	18.00	13.62	11.80	10.22	9.32			4.3	12.6	10.1	11.9	7.4	7.3
	Occipital	3.78	11.78	7.34	7.32	8.12	5.88			4.7	7.8	3.9	6.3	4.1	4.8
En	Frontal	4.82	23.76	21.54	14.14	14.62	11.60	6	3	2.1	10.2	7.2	6.3	5.9	5.4
	Motor	4.42	11.88	11.92	7.12	7.68	6.42			1.7	5.3	4.2	2.8	3.6	2.2
	Occipital	4.46	13.02	11.18	7.58	9.52	7.36			2.0	5.2	2.6	2.0	2.1	2.8

TABLE 3

THE MEAN PER CENT BETA ACTIVITY FOR THE PATIENTS WITH SCHIZOPHRENIA AND DEPRESSION BEFORE AND AT VARIOUS TIMES AFTER THE INJECTION OF THE DRUGS FOR EACH OF THE FIVE CONSECUTIVE DAYS AND FOLLOWING ELECTRIC CONVULSIVE THERAPY

		Schizophrenia						Depression					
			Minutes after injection						Minutes after injection				
Area	Day	Pre-injection	5	15	25	35	45	Pre-injection	5	15	25	35	45
Frontal	1	7.62	38.10	28.44	24.36	25.38	21.94	8.68	35.38	27.30	20.25	19.45	16.73
	2	8.02	36.72	29.20	21.36	17.46	14.78	7.40	34.18	26.73	19.50	19.73	17.80
	3	13.56	49.06	33.58	26.14	23.64	22.66	11.38	38.08	33.95	25.85	24.05	22.85
	4	11.26	36.66	26.94	22.34	19.36	14.86	8.50	33.80	22.55	22.45	20.80	13.15
	5	12.34	45.16	36.14	29.74	23.10	20.74	8.53	36.45	28.25	26.88	25.70	19.75
Pre-shock mean		10.56	41.14	30.86	24.79	21.79	19.00	8.89	35.58	27.76	22.98	21.94	18.05
Post-shock mean ...		4.60	9.64	8.86	7.22	7.64	7.32	4.05	14.68	11.85	9.95	8.27	6.85
Motor	1	6.76	25.54	20.54	17.72	17.62	16.90	4.85	29.00	22.58	18.75	15.95	14.20
	2	5.28	26.24	18.36	14.74	12.68	11.88	5.00	23.28	18.98	15.15	16.95	14.98
	3	9.40	34.70	26.92	23.12	19.64	18.64	7.05	30.60	25.18	22.85	20.58	19.65
	4	6.72	25.52	19.90	15.86	14.74	12.90	4.10	23.60	19.58	16.28	17.63	12.13
	5	9.52	32.64	29.72	24.58	18.96	17.94	5.98	27.43	22.98	20.23	18.90	18.90
Pre-shock mean		7.54	28.93	23.09	19.20	16.73	15.65	5.39	26.78	21.86	18.65	18.00	15.97
Post-shock mean ...		3.26	10.60	8.72	8.42	8.38	7.58	5.82	17.45	15.07	14.92	13.25	10.47
Occipital	1	8.74	19.20	14.28	15.02	12.04	13.30	3.60	14.70	9.10	9.15	8.10	6.15
	2	6.88	16.00	13.64	11.06	8.96	8.60	3.38	12.58	9.85	6.13	6.63	4.20
	3	12.94	28.30	21.50	18.20	15.82	15.90	4.85	15.90	11.78	9.60	8.60	7.23
	4	9.32	18.12	15.50	12.54	11.26	10.14	2.88	12.90	7.90	5.73	7.43	5.23
	5	10.96	26.64	24.00	18.86	15.38	15.10	4.78	13.68	10.38	7.68	9.85	7.80
Pre-shock mean		9.77	21.65	17.78	15.13	12.69	12.61	3.89	13.95	9.80	7.66	8.12	6.12
Post-shock mean ...		4.26	5.40	4.86	4.76	5.80	4.74	2.48	4.75	2.85	2.92	2.48	2.70

TABLE 4

CHANGES IN BETA ACTIVITY AND PSYCHIATRIC STATUS AFTER ELECTRIC CONVULSIVE THERAPY

	Schizophrenia					Depression				
	Difference in pre-injection beta (pre-shock minus post-shock)					Difference in pre-injection beta (pre-shock minus post-shock)				
Case	Frontal	Motor	Occipital	Status		Case	Frontal	Motor	Occipital	Status
Bu	1.16	2.16	0.80	Unimp.						
Jo	6.10	—1.90	—1.86	Unimp.		Pe	8.74	—2.26	2.36	Soc. Rec.
Pe	16.56	14.38	19.18	Soc. Rec.		Mz	4.84	—0.44	1.62	Rec.
Ne	—1.80	0.60	1.14	Soc. Rec.		Bl	3.08	—1.72	0.92	Rec.
Ba	7.78	6.14	8.28	Unimp.		En	2.72	2.72	2.46	Unimp.
Mean	5.96	4.28	5.51			Mean	4.84	—0.42	1.38	

TABLE 5

CLINICAL RESPONSE OF PATIENTS WITH SCHIZOPHRENIA AND DEPRESSION FOR FIVE CONSECUTIVE DAYS TO THE INJECTION OF THE DRUGS AND AFTER ELECTRIC CONVULSIVE THERAPY

	Schizophrenia						Depression						
	Day							Day					
Case	1st	2d	3d	4th	5th	Post-therapy	Case	1st	2d	3d	4th	5th	Post-therapy
Bu	M	M	M	P	P	P	..	..	..	..	..	..	..
Jo	P	P	P	P	P	P	Pe	G	G	G	G	G	P
Pe	M	M	P	P	P	P	Mz	P	P	P	P	P	M
Ne	M	P	P	P	P	P	Bl	M	M	M	M	M	P
Ba	M	M	M	P	P	P	En	G	M	M	M	M	M

G indicates a good reaction; M, a moderately good reaction; and P, a poor reaction.

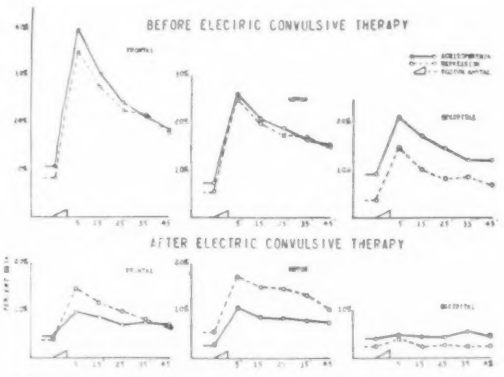

FIG. 1.—Mean per cent beta activity after injection of sodium amytal, at various intervals, in frontal, motor, and occipital areas, before and after electric convulsive therapy for patients with schizophrenia and depression. Upper half, before shock; lower half, after shock. (For method, see text.)

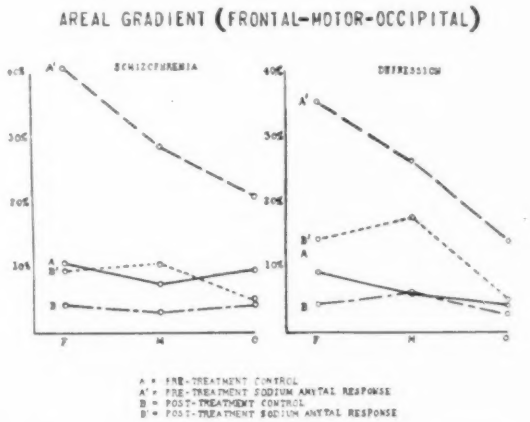

FIG. 2.—The areal beta-activity gradient (frontal-motor-occipital) before and after electric convulsive therapy. Gradients A and B are before and after the therapy respectively. Gradient A′ is the mean maximum (at 5 min.) per cent of fast activity after the injection of sodium amytal, before therapy; Gradient B′ the same, after therapy.

seen that no "tolerance" developed (Fig. 3, Table 3). In other words, neither the group with schizophrenia nor the group with depression evidenced any systematic decrease in beta with repeated administration of sodium amytal. This lack of significant electrocortical change stands in striking contrast to the psychologic tolerance which is developed by the schizophrenic patients (Fig. 3, Table 5).

After electric convulsive therapy, the per cent time beta in the frontal and occipital areas became markedly reduced for all patients in both groups (Figs. 1 and 2, and Tables 1, 2, and 3). The exception was the motor area in the depressed group. This reduction in fast activity not only appeared

in the control run but was made particularly prominent as a decreased responsiveness of beta activity following administration of sodium amytal (Fig. 2). Electric convulsive therapy, furthermore, had the effect of destroying the normal areal gradient, the destruction becoming exaggerated after the administration of the drug. This was clearest, and most dramatic, relative to the frontal area. In the schizophrenic patients,

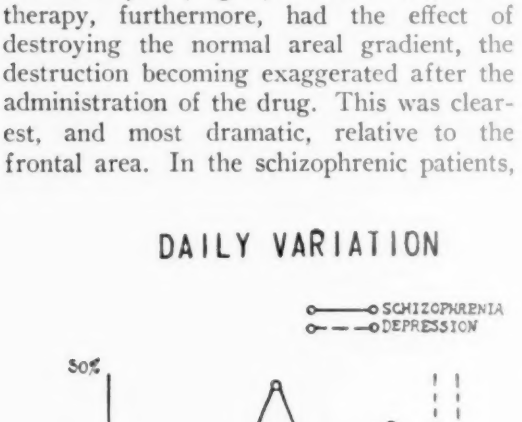

FIG. 3.—Daily variation (5 consecutive days) per cent beta activity (frontal area—5 min.) and clinical response to sodium amytal before and following electric convulsive therapy.

sodium amytal, after treatment, induced only slight increases in per cent time beta in the frontal and motor areas (these being equal) and induced no increase in the occipital area. The depressed patients exhibited post-amytal increases in the frontal and motor areas (motor slightly greater than frontal) and no increase in the occipital area. (Qualitative and quantitative inspection of the raw data indicate that this decrease cannot readily be accounted for in terms of masking of fast by slow activity(6), the latter frequently being increased by the convulsive therapies.)

Since electric convulsive therapy appeared to affect beta activity, both in the control and experimental periods, it seemed pertinent to discover whether or not the effect was reversible. From inspection of the published data of Hoagland, Malamud, Kaufman, and Pincus(1), we came to the conclusion that this indeed might be a variable worthy of isolation. In our own schizophrenic group, where the rate of administration of electric shock as well as total number of convulsions was, with one exception, held constant, it was possible to evaluate time after last treatment as a factor influencing beta and its response to sodium amytal. The data on the 5 patients, frontal area, gave a rank order correlation (rho) between time after last shock (range, 5 to 10 days) and the difference between pre-shock and post-shock beta response to sodium amytal of -0.82 (inferred $r = -0.83 \pm 0.1$). This indicates that in this group, at least, the greater the time since the last treatment, the more closely the beta response approaches the original level. Since N was so small, however, this coefficient should not be regarded as indicative, but as provocative.

We were unable to evaluate adequately the relationship between the number of convulsions and the beta activity, before or after the administration of sodium amytal, as the rate of administration of electric shock was different for the 2 groups, and there was insufficient range for adequate ranking.

There appeared to be no relationship between ratings of therapeutic efficacy (*recovered, socially recovered, improved, unimproved*) and post-shock per cent beta activity before or after amytal. Rank order correlations between per cent beta response to amytal, post-treatment, for the frontal area, and relative recovery, and between per cent beta, post-treatment, pre-amytal, for the occipital area, and relative recovery, were both less than 0.22; these are insignificant; the correlation apparently is zero. Likewise, rank order correlations between the difference in per cent beta pre- and post-treatment for frontal, motor, and occipital areas and relative recovery were insignificant (Table 4).

Discussion

These data may be discussed under several headings.

First, it is to be noted that the data provide further confirmation of the original disclosures of Cohn and Katzenelbogen(2), Rubin, Malamud, and Hope(3), and Brazier and Finesinger(4) concerning beta-response to sodium amytal and its areal gradient. The present data add the further information that, at least over a period of 5 consecutive days, there is no apparent systematic electrocortical tolerance developed to sodium amytal, as judged by the response selected for study.

Secondly, we feel that it is of definite interest to note that while there was stability of the beta-response to sodium amytal it was possible for the schizophrenic group to develop their characteristic tolerance in psychologic response to this drug. Thus, it may be supposed, on the basis of the patients studied here, that there is no universal, uncomplicated, one-to-one relationship between beta-response and psychologic improvement after injection of sodium amytal. The beta-response, one would infer from the Brazier and Finesinger(4) study, is a primary physiologic response; these data suggest that the psychologic response probably involves mechanisms other than those producing beta which may be differentially adapted in depression and schizophrenia. Indeed, Rubin, Malamud, and Hope(3) have indicated that, in schizophrenia, while beta activity increased with a decrease in "tension" after the injection of sodium amytal, there was a lack of relationship between other psychological effects and alterations in the EEG.

Third, our data indicate that for all cases studied, regardless of symptomatology, there was a marked and significant reduction of beta activity by electric convulsive therapy. Hoagland, Malamud, Kaufman, and Pincus(1) have previously reported a similar reduction in beta (or at least in frequency bands including beta) concurrent with electric convulsive therapy, although their data are limited to the occipital lead. They feel that the decrease in beta activity is related to improvement in psychiatric status. "When the patients improve, the percentage of time the waves are present decreases; when they relapse, the fast waves tend to return." When their published data are inspected from a viewpoint such as ours (which implies that reduction of fast electroencephalographic frequencies is a function of treatment rather than psychiatric status) it appears that, of 7 cases followed a sufficient length of time for inspection of the averaged data (10 days per point plotted graphically) to yield information, 4 cases showed quite clear increases in beta with the passing of time after shock, although all 4 continued to exhibit clinical improvement. One other case showed an increase in beta near the end of the treatment period, while shock was continuing, even though there was slight continued psychiatric improvement. Another showed continued reduction of beta, but great vacillation in psychiatric status. The remaining case, while exhibiting markedly reduced beta compared to the pre-treatment level, increased during the last half of the therapeutic period, and this rise was latterly associated with a rise in number of symptoms presented. Their actual data, therefore, are interpretable as indicating that decrease in fact is the consequence of shock.

The findings presented here rather strongly suggest that the reduction in beta is related to shock, *per se*. No relationship appeared between reduction in beta activity and post-shock improvement in mental status, although we would be the first to point out that our ratings of improvement were simpler than those of Hoagland, *et al*(1), whose new scale has much to commend it. Nevertheless, our quantitative electroencephalographic measures, made within 10 days of the termination of therapy, should offer evidence to distinguish between even qualitatively "improved" and "unimproved" patients.

Fourth, it would appear, in terms of the shift in the areal gradient reported here, as well as in view of increase of slow activity areally, that electric shock therapy has a relatively specific effect on anterior areas of the cerebrum. We do not know whether this is a characteristic of the areas, *per se*, or whether it is because, due to conventional electrode placement, anterior areas receive the greatest density of current flow. If pos-

terior placements were used, other shifts in the gradient might appear. It would be of interest also to discover whether a similar decrement follows prefrontal leucotomy.

Fifth, this primary change in beta activity, induced by shock, shows a further effect in the shift of the beta response to sodium amytal. The normal gradient is not only altered but the relative increase in beta for any area in response to amytal becomes restricted. This would seem to indicate that shock has greatly altered certain cerebral mechanisms represented electrically as beta frequencies. What operates to induce the shift of gradient and to prevent the response to sodium amytal, we do not know; nor do we know how many times current must flow through the frontal pole to produce the change; nor do we know the effect of increased number of treatments. In our own study, the schizophrenic group received twice the number that the depressed group received and at a faster rate. Their gradient was the most depressed, especially in response to amytal. The data Hoagland and his collaborators present would suggest that the alteration of per cent fast activity may be rather rapid, appearing after relatively few treatments.

From these data and their elaborations, we are led to the following tentative formulation:

1. Beta frequencies in the EEG appear distributed on an antero-posterior gradient.

2. Beta frequencies and their gradient are enhanced by sodium amytal.

3. Beta frequencies and their gradient are depressed by electric convulsive therapy (and the depressant effect probably includes the absolute number of shocks, and the time in days since last shock.)

4. The enhancement of beta activity by sodium amytal is less apparent after electric convulsive therapy.

This may be expressed in the form of an equation:

$$(A)\ \% \beta = f\left(\text{area, drug, } \frac{1}{N \text{ shocks}}, {}^{T} \text{ last shock}\right)$$

This implies that sodium amytal (barbiturates (4)) and electric convulsive therapy act in opposite ways on a common mechanism producing beta.

The psychologic changes induced by sodium amytal and electric convulsive therapy tend to act in a similar direction.

1. Psychologic state (ψ) is "more normal" after sodium amytal.

2. Psychologic state is "more normal" (but of a different quality?) after electric convulsive therapy.

Expressed as an equation:

$$(B)\ \psi = f\ (\text{drug, shock})$$

One hypothesis to account for these data would be that the neural mechanisms underlying beta and those underlying alterations in psychologic states are not identical. Further, definitive experimental effort is indicated to interrelate these mechanisms.

Summary and Conclusions

1. Nine patients, 4 with depression, 5 with schizophrenia, were studied with respect to changes in the beta activity of the EEG induced by the intravenous injection of sodium amytal mg. 250 (with racemic amphetamine sulfate mg. 20). These changes were measured on 5 consecutive days prior to a course of electric convulsive therapy. They were again studied in a single session, after this course of treatment.

2. There was observed to be a striking increase in beta activity after the injection. This increase was greatest in the frontal EEG, least in the occipital EEG, forming an anterior-posterior per cent time gradient, which was an accentuation of the preinjection gradient.

3. There was no systematic electrocortical tolerance developed in either group, to the drug, over a 5-day period. The schizophrenic group, however, developed the usual psychological tolerance.

4. After a course of electric convulsive therapy there was a decrease in percent time beta; the response of beta to sodium amytal was greatly reduced, particularly in the frontal area.

5. These data suggest that the electrocortical response measured (per cent time beta) and the psychologic state are reflections of the activity of different neural systems.

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DISCUSSION

ROBERT S. SCHWAB, M. D. (Boston, Mass.).—The wide use of electroshock therapy in the treatment of all kinds of psychiatric illness during the last 6 years has not produced very convincing evidence as to how this treatment produces its beneficial results. There have been some disturbing reports of neuropathological changes following electric shock therapy, demonstrated in the postmortems on persons that had died, presumably of other causes. There is also a good deal of animal postmortem tissue which fails to demonstrate any changes of a permanent nature in the brains, following shock treatment. This paper is, therefore, very important, in that it quantitates physiological changes following the use of electric shock treatment.

Before discussing in detail the results obtained, I would like to ask the speaker if the presence of benzedrine sulfate intravenously, in this large dosage that he uses—namely, 20 mgms., introduces any factors, in his opinion, that would modify the EEG. The paper by Rubin, Malamud, and Hope, quoted by the authors in their bibliography, reported that benzedrine sulfate tended to increase the amount of alpha activity in contrast to amytal, which reduced it and brought out the beta activity. I should therefore like to know whether they have some observations using the same comparative technique just described, where amytal was used alone, without the concomitant injection of benzedrine.

We have found, for instance, in assaying the effect of such a drug as neostigmin on various physiological functions in normal controls and in patients, that our early work showed some confusion of data, because of the presence of atropine, which was specifically added to prevent hypermobility of the intestinal tract. This did allow us, on the other hand, to use larger doses of neostimin without upsetting the subject. We finally concluded that the most reliable results were obtained when only one drug was injected. Indeed, the presence of benzedrine allows a larger amount of amytal to be used without inducing drowsiness or sleep. On the other hand, in addition to its central stimulating effect, benzedrine produces some relaxation in the smooth muscles and some evidence of sympathetic stimulation in the periphery and introduces, therefore, a conflicting factor into these experiments.

The second point refers to the use of monopolar leads. They probably do not reflect the same areal distribution found with bipolar leads, and this may explain the difference in this work from the clearer areal distribution in Brazier and Finesinger's work.

The other point that I should like to make is that the data on the persistence of the electroencephalographic changes described are not clear to me. In other words, the shock treatments may produce a *temporary* alteration in the neurophysiological state of the cerebral cortex, so that its response to the injections of amytal is changed from that before the electric shock, but after 3 to 4 months, this change may be completely reversible. There has been some evidence that the early success in leucotomies, for example, in reducing hypertension, was only of a temporary nature and that, 3 to 4 months after the operation, the blood pressure was again elevated. It is very important to know whether these physiological changes described here are due to permanent changes in structure and function or whether they are the temporary results of edema or injury. Of course, we do not insist on changes that last through the years, but I should be very much interested to know whether any of these subjects have been retested, 2 or 3 months after the electric shock treatment and what the result of this testing is. Previous work by the same authors showed that, for instance, in schizophrenics, the alteration after the first shock was quite different from that after the last shock, a fact which they explained by the endogenous adaptability of the schizophrenic patient. It is well known that neuropathological changes produced by a cerebral trauma, anoxia, and therefore, of course, by electrical shock, when they are completely reversible, may take several months to go through this process. Therefore, I think it is essential, before drawing conclusions as to the meaning of these neuropathological changes, that we know something about their survival.

Brazier and Finesinger, working in our laboratory at the Massachusetts General, have shown interesting data on the return of the cerebral electrical activity, after injections of amytal, to the pre-injection level. I note from Table 1 in the

manuscript that the return to the pre-injection level here was only partial after 45 minutes, whereas in their work it was nearly back to normal in 40 minutes and completely back in 80 minutes. I am wondering whether benzedrine would delay the returning of the EEG after the injection to its former level. Furthermore, the peak of the beta activity in Brazier and Finesinger's work appeared 20 minutes after the injection; the peak in this series seems to be much earlier—namely, 5 minutes after the injection. I am wondering what the author will have to say about this.

I believe all this work shows that it is erroneous to suppose that the presence of 10 per second alpha activity is always associated with calm and placid individuals, because here we have evidence that the presence of the faster beta rhythms on one hand is associated with reduction in anxiety and improvement of the condition following the injection of amytal, and obversely clinical improvement over a longer time occurs when this beta wave response to amytal is greatly reduced.

J. S. Gottlieb, M. D.—Dr. Schwab, in his discussion, has raised some pertinent questions with reference to the data presented above. His questions, however, ask for information which may be answered only by the extension of the present studies. The addition of benzedrine sulfate to the sodium amytal no doubt modifies the EEG, as, for example, it has been known to modify the psychologic effects, but the response of the EEG to sodium amytal with and without benzedrine sulfate constitutes a study of considerable magnitude.

As a matter of fact, in the opinion of the authors the present study is of importance for the questions it raises rather than for the answers it gives. Dr. Schwab has pointed out certain critical variables which might profitably be isolated for study: drugs; bipolar or monopolar leads; time as a factor in evaluation of residuals of electroshock. In addition to these, our attention has been focused on other variables which interest us: the exact placement of electrodes; the effect of area or region of application of shock (*i.e.*, is the change due to density of flow of current, or to susceptibility of a particular region?); the relation of EEG response to drug and number and frequency of electrically induced convulsions (*i.e.*, shifts in areal gradient, reduction in amytal response); the selection of patient populations (*i.e.*, are there differential EEG responses in different diagnostic groups?); the amount of drug or drugs. In addition, after defining these variables, it may be that there are therapeutic implications in the EEG drug response.

The study of these variables, irrespective of the fruitfulness of such an approach into the neurophysiologic mechanisms of electric convulsive therapy and possibly schizophrenia and depression, is complicated by the problem of quantification of EEG data. It is our hope that research in these areas will be stimulated by this report. Our own endeavors are now temporarily delayed awaiting the construction of a frequency analyzer (Grey Walter) which we hope will permit us to amplify our research.

CEREBROSPINAL FLUID CHANGES IN ELECTROSHOCK TREATMENT OF PSYCHOSES

(Spectrophotometric and Enzyme Studies)[1]

M. SPIEGEL-ADOLF, M. D., P. H. WILCOX, M. D., and E. A. SPIEGEL, M. D.

Previous studies (Spiegel-Adolf, Spiegel, Ashkenaz, and Lee(1)) have demonstrated that the cerebrospinal fluid (CSF) of dogs subjected to electrically induced convulsions showed band absorption between wave lengths 2550 and 2850 Å with a peak at 2650 Å. Since abnormal protein content could be excluded and barbiturates had not been administered, it was inferred that this absorption band was caused at least partly by nucleic acids or their constituents belonging to the pyrimidine-purine group, and that a breakdown of nuclear substances takes place under the influence of the convulsions.

In view of the scarcity of our knowledge of the mechanisms underlying the therapeutic effects of electroshock, it seemed of interest to extend these studies to psychotic patients subjected to electroshock treatment.

Material and Method.—The material consisted of 32 schizophrenics, 16 of whom had not received electroshock treatment (ECT) previously, of 10 psychotics having schizoid components, and of 4 low-grade mental defectives. A total of 105 CSF's were studied. The CSF's of 35 patients without neuropsychiatric disorders analyzed in a parallel study of cerebral concussion served as controls. Barbiturates were avoided since the appearance of such drugs in the CSF gives selective absorption similar to the one caused by nucleic acids. All CSF's used were clear and colorless and were studied within ½-1 hour after the tap.[2] During this interval they were kept on ice. For study of the absorption spectrum in ultraviolet light, a Beckman photoelectric quartz spectrophotometer with a 5-mm. cell was used. Saline solution served for comparison. The spectrophotometric analysis was made between 2200 and 3100 Å, in steps of 10 Å. The extinction coefficient for each reading was computed in the usual manner, and a graph was plotted for each sample. The concentration of the undiluted CSF was arbitrarily chosen as the unit. The absorption at 2650 Å was computed from the spectrophotometric graphs. Routine examination of cell count, protein content, colloid gold reactions were performed in parallel samples by the clinical laboratory. Other methods used in this study will be described later on in this paper.

Results

The Cerebrospinal Fluid in Untreated Schizophrenics.—The absorption spectrum of a cerebrospinal fluid from a neurologically normal individual, without destructive processes in other tissues, and without barbiturate medication, gives an **S**-shape graph (Fig. 1). In contradistinction, the CSF's of untreated schizophrenics show marked selective absorption at 2650 Å. (Table 1, Fig. 1).

This table shows that the mean extinction coefficient of our untreated schizophrenics was E (2650) = 1.99 ± 0.5, while in 28 control cases studied by means of the Beckman spectrophotometer [3] the mean E (2650) = 1.24 ± 0.59. The difference between the mean of the schizophrenics and that of the normal controls proved statistically significant (t = 4.47: see footnote to Table 1). It should be pointed out that the routine examination of

[1] Read at the 103d annual meeting of The American Psychiatric Association, New York, N. Y., May 19-23, 1947.

From the Traverse City State Hospital, Traverse City, Michigan, and the Departments of Colloid Chemistry and Experimental Neurology, Temple University School of Medicine, Philadelphia, Pa.

Aided by a grant from the Electroshock Research Association.

[2] As a rule 2 samples of 7 cc. each were withdrawn; the first sample was used for routine examination (proteins, cells, Wassermann, colloidal gold), the second for the spectrophotometric and enzyme studies. It was assumed that this second sample originating from higher parts of the subarachnoid space was preferable for a study of metabolic products originating in the cerebrum.

[3] The other control cases were studied by means of a Hilger quartz spectrograph and showed plainly a lack of selective absorption.

the CSF's of these untreated schizophrenics failed to detect pathological changes; this is in agreement with the findings of Merritt and Fremont-Smith(2).

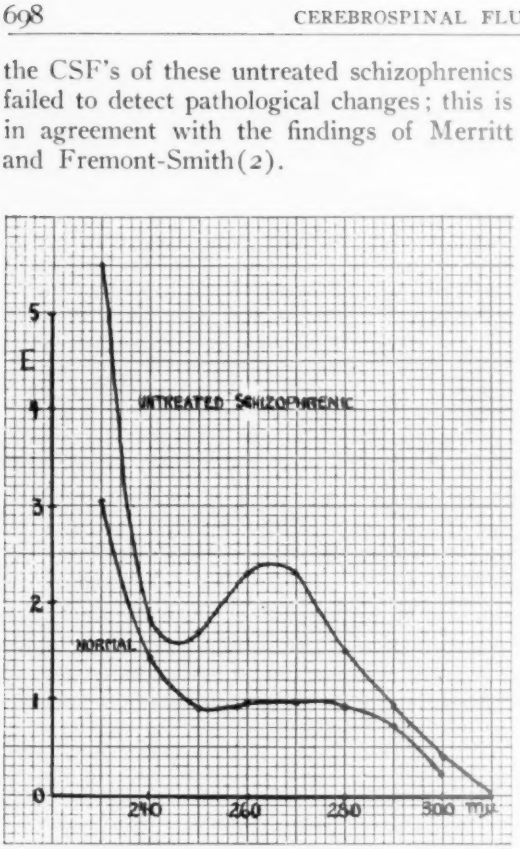


FIG. 1.—Absorption graph of a neurologically normal subject and of an untreated schizophrenic. E = extinction coefficient.

TABLE 1

Mean extinction coefficient E at 2650 Å		
Untreated schizophrenics	1.99 ± 0.5	(M_s)
Cerebral concussion	2.47 ± 0.73	(M_c)
Control cases (without organic diseases of the CNS or psychosis)	1.24 ± 0.59	(M_n)
$M_s - M_n$ statistically significant	($t = 4.47$ *)	

* t = difference between the means divided by the standard error of the difference (see Fisher(1a), p. 120).

A deviation from the normal in the CSF of schizophrenics was observed by Spiegel-Adolf and Freed(3) who determined the ratio of nonelectrolytes to electrolytes. For untreated schizophrenics this ratio was 0.26, while in neurologically normal cases Spiegel-Adolf(4) found an average value of 0.22. Thus physicochemical methods, such as determination of the ratio of nonelectrolytes to electrolytes and spectrophotometry point to changes deviating from the normal composition of the CSF.

It seems of interest to compare these data with the results of a parallel study of cerebrospinal fluids in cerebral concussion(5). In a group of such patients tapped between 1 hour and 2 months after the accident, we obtained E (2650) = 2.47 ± .73. The fact that following mechanical trauma to the brain the selective absorption of the CSF at 2650 Å is still more pronounced indicates that our findings in the CSF of schizophrenics are not specific for this disease. Our observations on the CSF's of low-grade mental defectives point in a similar direction. For this group the values of E (2650) varied between 3.1 and 3.6. Thus it seems that a high selective absorption at 2650 Å is a nonspecific sign of pathologic changes in the central nervous system, similar to other characteristics of the CSF. In the concussion study it was pointed out that the high selective absorption at 2650 may be a correlate of chromatolytic changes in the nerve cells; other possibilities are injury to nuclei directly or anomalies in the vitamin C metabolism.[4] Further studies will have to elucidate these problems.

Effect of Electrically Induced Convulsions.—Two types of electrical stimulation were used: a relatively high (600 ma ± 50 ma) 60-cycle A. C. for 0.4 to 0.6 second with the electrodes applied in bitemporofrontal posi-

[4] It is well known that ascorbic acid in the CSF also gives a selective absorption at 2650 Å as pointed out by one of us (Spiegel-Adolf) previously (5a). After this paper went to press, Strait *et al.* (5b) tried to prove that the selective absorption of the CSF at this wave length is due to ascorbic acid only. Their method of assay leads to the paradoxic result that the concentration of ascorbic acid in the CSF is twice that in the blood. They overlook that there is an equilibrium between blood and CSF, and that no other known constituent of the CSF reaches a higher concentration than 20-30% above the blood level. A concentration of ascorbic acid in the CSF corresponding to the generally accepted blood level (up to 2 mg per 100 cc.) would give, according to unpublished measurements of one of us (Spiegel-Adolf), an E(2650) within the range of the values found in our normal controls. Furthermore, Strait *et al.* seem to ignore that barbiturates entering the CSF give also an absorption in the neighborhood of 2600 and that such a medication must therefore be stopped preceding such measurements. Finally they disregard that the existence of nucleic acid splitting substances in the CSF was demonstrated(6) by studying the fate of nucleic acids incubated with CSF.

tion (Cerletti-Bini technique) or a low intensity current (85 ma ± 10 ma, modified half wave rectified 60-cycle current, Reiter technique). The latter was applied for 0.6 to 0.8 interrupted second (commutator on 10 times a second and off 10 times a second); the electrodes were in vertex positive left temple negative electrode position.

After application of the high 60-cycle A. C. current, there was in the beginning of the series (after 3-6 convulsions) a slight depression of the E values (Figs. 2 and 3) and

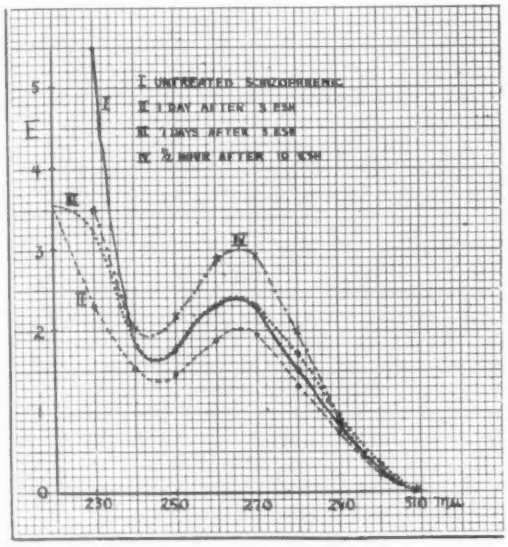


FIG. 2.—Effect of electroshock treatment (Cerletti-Bini method) upon absorption graphs of a schizophrenic. E = extinction coefficient.

only occasionally a rise or no change. At the end of the series (after 10 convulsions) an increase of the E value developed in 8 out of 9 cases (Table 2), which proved statistically significant.

A further group (No. II) of 6 chronic women may here be added. They had received from 4 to 67 ECT's (mostly Reiter type) none of which more recently than 4 months before this series. The CSF was studied 1 day after 7 high AC ECT's. It showed E (2650) values ranging from 0.76 to 1.46. Three of these patients were retapped after an interval of 3 weeks without further ECT and showed a rise of the E values (from 0.76 to 1.4, from 0.95 to 1.4, from 0.92 to 1.9).

Following 3-10 convulsions induced by the low intensity current, there was in the majority of the observations a slight decrease (Fig. 4) or no change of the E value

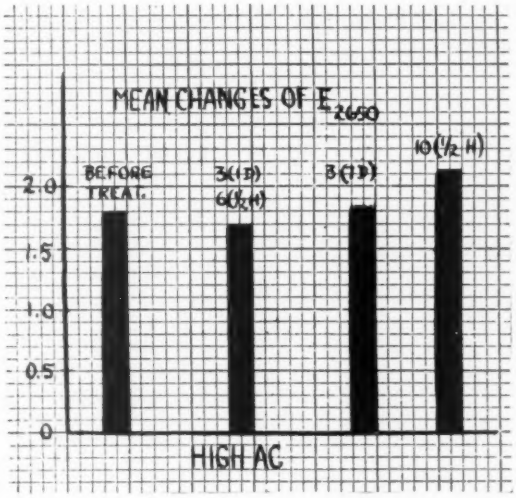


FIG. 3.—Mean changes of E at 2650 Å following electroshock treatment (Cerletti-Bini method).

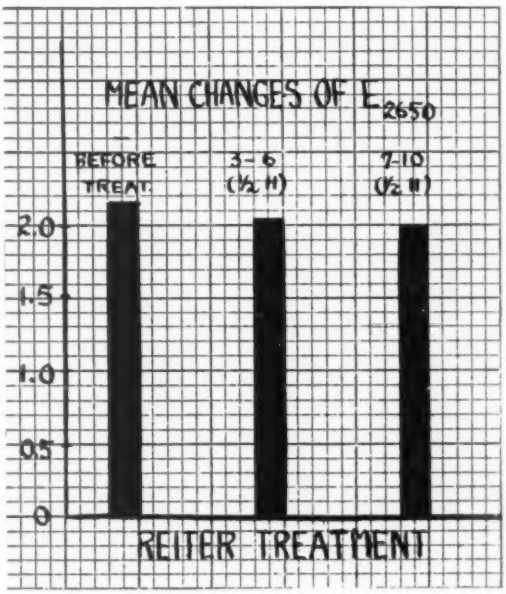


FIG. 4.—Mean changes of E at 2650 Å following electroshock treatment (Reiter method).

and only in few cases a slight increase of the selective absorption as shown by Table 3. These changes were of questionable statistical significance. Further studies will have to ascertain whether application of low inten-

sity current for more prolonged periods may produce a similar rise of E_{2650} as observed after repeated ECT with high AC.

In order to elucidate the mechanism of these changes the enzymatic power of these CSF's upon samples of nucleic acids of plant origin (yeast type, Schwarz), as well as of animal origin (thymonucleic acid type, Difco)[5] was tested, similarly as in our previous studies of cerebral concussion(6). Samples of the CSF's were mixed with the nucleic acid (Nu) preparation under study, and this mixture as well as CSF alone in corresponding dilution, and the solution of the nucleic acid alone were incubated for 4 hours and then submitted to spectrophotometry. For all 3 solutions (CSF alone, nucleic acid solution alone, CSF—nucleic acid mixture) the extinction coefficient E was determined at 2600 Å.[6] The ability of the CSF to split nucleic acids was determined according to formula $x = E(Nu) + E(CSF) - E(CSF - Nu)$. In Table 4 x is computed for 100 cc. CSF.

TABLE 2

EFFECT OF HIGH INTENSITY A. C.

Extinction coefficient at 2650 Å

Patient No.	A Before treatment	B	C	D	D-A
		After treatment with high 60-cycle A. C.			
Chronic untreated men (Group I)					
1	2.4	1.94 1 day III	2.3 7 days III	3.0 30 min. X	0.6
2	2.2	2.26 1 day III	2.1 7 days III	2.85 30 min. X	0.65
3 (1 ECT 2 mon. previously)	1.35	1.26 1 day III	1.55 7 days III	1.75 30 min. X	0.4
4	1.9		1.45 7 days III	2.1 30 min. X	0.2
5	1.8	2.49 1 day III	2.10 7 days III	2.6 30 min. X	0.8
6	1.1	1.25 1 day III	1.45 7 days III	1.25 30 min. X	0.15
Chronic treated women (Group V)					
32	2.2	1.6 30 min. VI		1.8 45 min. X	−0.4
33	1.65	1.6 15 min. VI		2.0 15 min. X	0.35
34	1.6	1.5 30 min. VI		1.9 30 min. X	0.3
	Mean A = 1.80	Mean B = 1.74	Mean C = 1.82	Mean D = 2.13	Mean D-A = .34 (= 18.8% increase) $t^* = 2.9$ ($P < 5\%$)

Explanation: Time = interval between last convulsion and spinal tap.
Roman numeral indicates number of treatments.

* t = mean divided by standard error of the mean (see Fisher(1a), p. 117).

[5] Nucleic acid of the ribose type (yeast type) prevails in the cytoplasm (particularly Nissl bodies); nucleic acid of the thymonucleic acid type (desoxyribose type) occurs in the nucleus of cells only (Greenstein(6a)).

[6] The peak of the selective absorption of the CSF lies at about 2650 Å, while the peak for nucleic acids (*e.g.*, of pure thymonucleic acid) lies at 2600 Å. It seems that a shift of the peak to longer wave lengths occurs for compounds of nucleic acids and proteins (Lavin, Loring and Stanley(7), Spiegel-Adolf and Wycis(8)). In agreement with this view, recent studies of Euler and Hogberg(9) on nucleoproteins and dissolved nuclei showed that desoxyribonucleic acid has an absorption maximum at 2610-2630 Å.

Table 4 shows that the CSF of untreated schizophrenics has the ability to split plant nucleic acids. The mean of 22.6 ± 21.9 compares with a value of 16.2 obtained for normal controls in a previous study on cerebral concussion (5). The difference between these means is of questionable significance. However, it may be of some significance that the values were particularly low in group I (untreated chronic men, Mean $A_I = 3.0$) and much higher in group IV (untreated acute women, Mean $A_{IV} = 30$) and in group VI (untreated acute men, Mean $A_{VI} = 34.6$); but more studies seem necessary to ascertain whether these differences between chronic and acute cases are a constant finding. In any case the high standard deviation seems of interest in view of Hoskins' (10) statement that the standard deviation of the mean, which is a measure of variability, is much higher in schizophrenics than in normals. For various metabolic functions,

TABLE 3

EFFECT OF TREATMENT WITH LOW INTENSITY CURRENT *

Extinction coefficient E at 2650 Å

Patient No.	A Before treatment	B After treatment	C After treatment	C-A	D After treatment
Group IV. Acute untreated women					
25	1.47	1.4 75 min. III	1.63 17 min. VII	.16	
26	2.37	2.1 1 day III	2.4 25 min. VII	.03	
27	1.81	1.76 1 day III	1.6 40 min. VII	— .21	1.8 4 days VII
28	2.4	bloody	2.15 30 min. VII	— .25	
30	2.3	2.41 1 day III	2.05 24 min. VII	— .25	
Group V. Chronic treated women					
31	2.1	1.7 17 min. VI	1.7 22 min. X	— .40	
35	2.1	2.2 15 min. II	1.9 37 min. VI	— .20	2.3 15 min. X
36	2.85	2.85 26 min. VI	2.7 19 min. X	— .15	
Mean A (IV + V) = 2.17			Mean C (IV, V) = 2.01	Mean A-C = — .16 ± .16 $t = 2.5$ significance doubtful	
Group VI. Acute untreated men					
49	1.9	1.9 16 min. V			
50	2.6	2.2 28 min. V			
51	2.6	2.3 23 min. V			
52	2.95	2.82 32 min. V			
53	1.6	1.7 43 min. V			
54	1.25	1.25 53 min. V			
Mean A (IV, V, VI) = 2.16		Mean B = 2.04			

Explanation: Time = interval between last convulsion and spinal tap.
Roman numeral indicates number of treatments.

* In groups IV and VI low intensity rectified 60-cycle current was applied, in group V low A. C. was used (200 ma ± 10 ma, 0.8 to 1.2 sec.).

Hoskins finds the average variability in schizophrenia from 1½ to twice that in normal persons of similar age.

Following electrically induced convulsions, we find in the beginning of the series (after 3 to 5 treatments) a distinct increase of the splitting activity of the CSF upon plant nucleic acids (in 14 out of 15 cases). Only in case number 53 was there a decrease of the value. The difference between the mean B (after 3 or 5 treatments) and mean A (before treatments) proved statistically significant. In the further course of the treatment, however, the enzymatic ability of the CSF's again decreases (Fig. 5). While the values are still definitely above the pretreatment level following 10 applications of high A. C., we find that following 7 Reiter treatments the mean values of the enzymatic activity are no longer significantly different from the pretreatment values.

In principle quite similar conditions have been found (Table 5; Fig. 5) regarding the effect of the CSF upon animal nucleic acids (thymo-nucleic acid type). The enzymatic activity of the CSF from untreated schizo-

TABLE 4

ENZYMATIC EFFECT OF CSF UPON PLANT NUCLEIC ACIDS (YEAST TYPE, SCHWARZ)

Decrease of extinction coefficient at 2600 Å produced by 100 cc. CSF

Case No.	A Before treatment	B After 3 high AC (I), 3R (IV), or 5R (VI)		B-A	B^1 1 week after B	C After 10 high AC (I) or 7R (IV)		D After 7R
Group I. Untreated chronic men								
1	0	0.16		0.16	120	22		
2	0	52		52	0	78		
3	2	140	(1d)	138	14	14	(¼-½ h)	
4	0	...		...	10	32		
5	16	88		72	4	22		
6	0	1.6		1.6	36	12		
	($MA_I = 3$)	($MB_I = 56.3$)			($MB'_I = 30.6$)	($MC_I = 30$)		
Group IV. Untreated acute women								
25	2	24 (½ h)		22		6		
26	0	60		58		2		
27	32	60		28		10	(½ h)	16 (4 d)
28	44	...	(1 d)	...		54		
29	60	...		...		..		
30	42	69		27		78		
	($MA_{IV} = 30$)	($MB_{IV} = 53.2$)				($MC_{IV} = 30$)		
Group VI. Untreated acute men								
49	42	54		12				
50	30	52		22				
51	34	60	(¼ h-¾ h)	26				
52	0	94		94				
53	62	32		−30				
54	40	58		18				
	($MA_{VI} = 34.6$)	($MB_{VI} = 58.3$)						
Mean	22.6 ± 21.9	56.3		36.1				
				$t = 3.4$ (P : 1%) significant				
Mean (except nos. 4, 28, 29) = 20.1								

Explanation: R = treatment with low intensity rectified current.
In brackets: Group No. in Roman numerals, time interval after last treatment in hours (h) or days (d).

phrenics (A) is lower than that upon the plant nucleic acids (12.4±12.0 compared to 22.6±21.9). Control cases studied in the concussion series gave a mean value of −1.3. Rise of this enzymatic activity following 3 or 5 treatments is less regular than in regard to the plant nucleic acids, since we find here only in 11 out of 15 cases an increase of the enzymatic activity, but there is still a signifi-

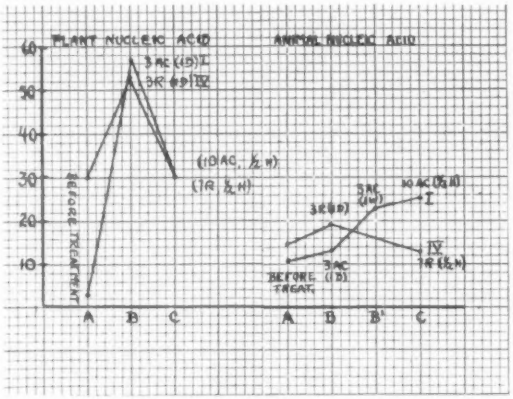


FIG. 5.—Enzymatic effect of CSF's upon nucleic acids. Decrease of extinction coefficient at 2600 Å. Mean values calculated for 100 cc. of CSF.

AC = high AC treatment.
R = Reiter.
D = day, H = hour, W = week } after last electroshock treatment.

cant increase of the mean. In group I the mean further increased after 1 week's interval (B^1). After continued treatment, we find that the mean value drops after 7 Reiter treatments (C_{IV}) while no definite trend is noticeable after 10 high AC applications in group I. Thus we find again that electroshock produces at least a transitory rise of the enzymatic activity.

In previous studies of cerebral concussion (11) we found in the CSF not only substances able to split in vitro nucleic acids and their constituents upon which the specific absorption in UV light depends, but also substances producing chromatolysis on anterior horn cells in sections from cats' spinal cords. It seemed of interest to ascertain whether similar substances are demonstrable in the CSF following electroshock. The technique was similar to that described in the last mentioned paper.

Paraffin sections 5 to 10 microns in thickness were prepared from cats' cords. Two pairs of such sections were placed on a slide. After the sections were deparaffinized, each pair was surrounded by a square frame corresponding to the size of a coverglass of 18 sq. mm. Within 1 hour after tapping, 0.45 cc. of the fluid was mixed with 0.05 cc. of an acetate buffer of pH 4.05 in a sterile tuberculin syringe. In later experiments also a mixture of 0.5 cc. CSF and 0.1 cc. buffer was used. About 0.2 to 0.25 cc. is necessary to fill the area within the paraffin square. After this amount of fluid has been deposited within the paraffin frame, it is covered by a clean cover glass, and the edges between the coverglass and the frame are sealed with paraffin so that they are air tight. It is important that the fluid cannot evaporate when the slide is put in the incubator and that no air bubbles are left within the sealed area, since changes in the concentration of the fluid may affect the nerve cells. Usually one pair of sections is covered with CSF and the other with Ringer's solution, which is also acidified with the buffer solution in the same proportion, 9 to 1, or 5 to 1. The same procedure may be repeated on the second slide, or on the second slide only one square is covered with CSF and the other square left without fluid. The slides are then put in an incubator at 37° C. for 4 hours. At the end of this period, the coverglasses and the paraffin frames are removed, and the slides are washed in distilled water and subjected to routine staining with 0.1 per cent thionine solution. On differentiation of the slides with alcohol, it is important to check the progress of the differentiation under the microscope repeatedly. If the CSF produces a high degree of tigrolysis, the differentiation should not be too prolonged, so that the cytoplasm of the cells is still clearly visible. In such a case the control sections which were under the influence of Ringer's solution or were not subjected to any fluid at all, are, of course, slightly overstained. In any case it is important to treat both pairs of sections, those under the influence of CSF and the control sections, in exactly the same manner.

As a rule the marked effects of the CSF upon anterior horn cells, as observed in our study of cerebral concussion, could not be found in the present series. The CSF of some of our concussion cases had been able to produce nearly complete tigrolysis and homogenization of the protoplasma in the anterior horn cells. In contradistinction, following incubation of the CSF's from patients who had received electroshock treatment, with sections of the spinal cord, tigroid bodies usually were still demonstrable in most anterior horn cells. However, quantitative differences could be found on comparison with control sections incubated with buffered Ringer's solution. The anterior horn cells of these control sections still showed

the typical coarse tigroid bodies, while in the cells exposed to the CSF's after electroshock the tigroid was represented by fine, lightly stained granula or a delicate network. In some instances (*e.g.*, sample No. 80 from case No. 2 in group I, after 10 high AC treatments (Fig. 6), No. 71 after 7 Reiter treatments in case 30, group IV) definite tigrolysis (usually starting from an area at the periphery of the cells) and occasionally cell shadows were found. In this connection it seems of interest that Windle, Krieg, and Arieff(12) found the tigroid structure intact following a single electroshock in guinea pigs, while concussion evoked definite chromatolysis. The effect of repeated electroshock apparently was not studied by Windle et al.; they are inclined to view the reports of ganglion cell disease following electroshock with skepticism ascribing them to inadequate fixation of the tissue. It seems that some of the findings of slight chromatolytic changes in nerve cells following repeated electroshock reported by experienced investigators familiar with post mortem changes, *e.g.*, those by Neubuerger, Whitehead, Rutledge, and Ebaugh(13), deserve consideration. Our findings that, following repeated electrically induced convulsions, substances able to affect the tigroid bodies diffuse into

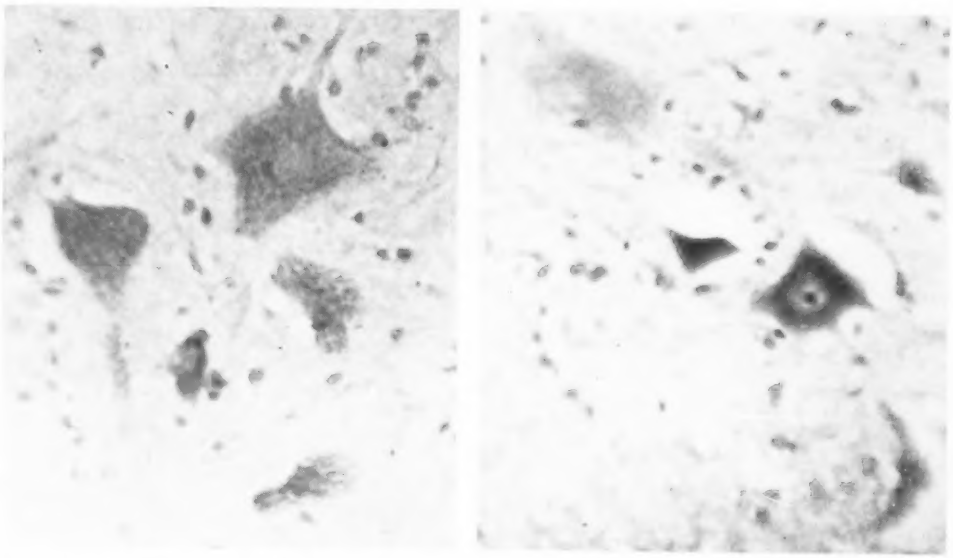
TABLE 5

ENZYMATIC EFFECT OF CSF UPON ANIMAL NUCLEIC ACIDS (THYMONUCLEIC ACID TYPE, DIFCO)

Decrease of extinction coefficient at 2600 Å produced by 100 cc. CSF

Case No.	A Before treatment	B After 3 high AC (I), 3R (IV), or 5R (VI)		B-A	B^1 1 week after B	C After 10 high AC (I) or 7R (IV)		D After 7R
Group I. Untreated chronic men								
1	0	0.4	1 d	0.4	22	34	½-½ h	
2	40	1		−39	16	40		
3	2	26		24	44	3.2		
4	20	...		...	30	28		
5	2	28		26	0	30		
6	0	4		4	28	14		
	($MA_I = 10.6$)	($MB_I = 11.9$)			($MB'_I = 23.2$)	($MC_I = 24.8$)		
Group IV. Untreated acute women								
25	6	40 (½ h)		34		8	½ h	40 (4 d)
26	0	12 (1 d)		12		8		
27	24	18 (1 d)		−6		8		
28	18	...		...		20		
29	20	...		...		..		
30	18	45 (1 d)		27		24		
	($MA_{IV} = 14.3$)	($MB_{IV} = 19.7$)				($MC_{IV} = 13.6$)		
Group VI. Untreated acute men								
49	28	16	¼-¾ h	−12				
50	5	36		29				
51	13.4	54		40.6				
52	0	44		44				
53	0	36		36				
54	26	22		−4				
	($MA_{VI} = 12$)	($MB_{VI} = 34.6$)						
Mean	12.4 ± 12.0	25.4		14.4				
				$t = 2.3$				
				Significant (P : 5%)				

Explanation: R = treatment with low intensity rectified current.
In brackets: Group No. in Roman numerals, time interval after last treatment in hours (h) or days (d).



A—after incubation with buffered Ringer's solution.

B—after incubation with cerebrospinal fluid No. 80, obtained after 10 high AC treatments.

FIG. 6.—Anterior horn cells of normal cat's spinal cord, stained by Nissl method.

the subarachnoid space seem to support the concept that severe and repeated electroshock may eventually produce at least transient structural changes in the nerve cells.

Since lipoids form one of the most important constituents of the central nervous system, it seemed of interest to ascertain whether an enzymatic activity or an influence upon such an activity could also be demonstrated regarding this group of substances. We have tried to estimate the content of the CSF of olive oil lipase and of ethylbutyrate esterase by using the method developed by Crandall and Cherry (14) and by Kaplan, Cohn, Levinson, and Stern(15). Table 6

TABLE 6

LIPOLYTIC ACTIVITY EXPRESSED IN MILLIMOLS OF FATTY ACID PER 100 CC. CSF MEAN VALUES

Untreated schizophrenics	0.075
After 3-5 Reiter treatments	0.18
After 10 high AC or 7 low rectified current.	0.36
Chronic treated schizophrenics (groups II, III, V)	0.36

summarizes these measurements giving the mean values of the lipolytic activity of the CSF's expressed in millimols of fatty acid per 100 cc. CSF.

As shown by Table 6 the lipolytic activity of the CSF in untreated schizophrenics is negligible. Following 3-5 Reiter treatments, and still more marked following 7 Reiter or 10 applications of high alternating current, somewhat higher values of lipase activity were found, and it is interesting that the same average value was obtained in the chronic treated cases. However, all these values are within the range considered by Kaplan et al.(15) as normal, although it should be emphasized that the normal values given by these authors were obtained on epileptics, cases of cerebral birth trauma, and idiocy. Only in some single instances values beyond the normal range were found in our material, for instance, in case numbers 16 (1.5), 18 (2.0), and 35 (2.0). An explanation of these relatively high values could not be found. In any case degenerative processes as far as they may be revealed by an increase of lipolytic activity of the CSF seem to take place only occasionally in untreated schizophrenics as well as following electroshock. This is in contradistinction to the distinct effect of electroshock upon the nucleic acid metabolism. The fact that our material shows an increase of substances apparently belonging in the groups of nucleases or desaminases [7] but not of lipases or ethylbutyrate esterases may be of interest for the following reason. The question may be raised whether the nucleases and desaminases appearing in the CSF in our cases derive from the blood or from the central nervous system. In order to answer this question it should be remembered that all enzymes are proteins and have a very high molecular weight. If the appearance of increased amounts of nucleases in the CSF were due to increased permeability of the blood-CSF barrier, one should expect to find increased protein content in the CSF. However, the protein values of these CSF's were as a rule within normal limits. Furthermore, one would expect that such an increase of permeability of the blood-CSF barrier would allow not only nucleases, but also lipases to enter in increased amounts from the blood into the CSF. Thus the absence of abnormal amounts of protein as well as of definitely increased lipolytic activity of the CSF seems to indicate that the increased amounts of nucleases found in our material are not due to increased permeability of the blood-CSF barrier, but that these substances derive from the tissue of the central nervous system.

The question may be raised whether the observations reported in this paper may help one to understand the obscure mechanisms responsible for the therapeutic effects of electrically induced convulsions. Recently Hoskins(10) has pointed out that one of the most characteristic features of schizophrenia is a deficiency in the uptake of oxygen, and he is inclined to attribute the defective oxygen assimilation to lack of enzymes or coenzymes. If this concept is correct, our observations that electroshock increases the activities of enzymes related to the nucleic acid metabolism of the brain may perhaps serve as an indicator that electro-

[7] It would be premature to identify the chemical nature of the enzymatic substances concerned. However one should bear in mind that they are able to split not only nucleic acids, but also their nitrogen-containing constituents upon which the specific absorption band depends. Thus one deals apparently not only with nucleases, but also with desaminases.

shock may have a similar effect upon other, more important, enzyme systems of the central nervous system. The possibility that such effects of electroshock may be a factor in the mechanism of this therapeutic procedure is offered as a working hypothesis only. Further studies are planned, in order to test its usefulness.

This paper does not attempt to cover the clinical changes occurring in patients as the result of these treatments, but the chronic schizophrenics who were subjected to 10 daily high A. C. (Cerletti-Bini technique) ECT's appeared worse (organic reaction pattern) than at the beginning of the series and no long-term gains were noted. Patients subjected to 7-10 daily Reiter treatments did not show this organic reaction pattern.

SUMMARY

1. The cerebrospinal fluid of untreated schizophrenics shows a pronounced selective absorption in ultraviolet at $\lambda = 2650$ Å.

2. Electroshock treatment influences the ultraviolet absorption of the cerebrospinal fluid (initial decrease, followed by increase on repeated application of high A. C.).

3. Enzymatic substances apparently belonging to the groups of nucleases and desaminases increase in the CSF following electroshock convulsions.

4. Lipases are much less affected by electroshock convulsions than are the nucleases.

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THE EFFECT OF ELECTRIC SHOCK ON MENTAL EFFICIENCY [1]

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Since the advent of shock therapies, persistent questions have been raised concerning the effect of shock on intellectual functioning. Clinically, electric shock produces an impairment of mental efficiency. This is of varying degrees of severity, ranging from "organic" syndromes characterized by confusion, disorientation, marked memory disturbance, and affective changes to conditions of slight memory loss(7). In general, the larger the number of shocks, the shorter the interval between shocks, and the older the patient, the greater the impairment(4). These effects are usually transitory so that 10 to 15 days after the last treatment there is little, if any, gross evidence of impairment except for complaints of slight memory difficulty and an amnesia for part or all of the period of shock treatment. After 6 to 8 weeks most of these complaints disappear, although some patients retain an amnesia for the period of shock treatment(8).

The absence of clinical evidence of residual intellectual impairment after a longer period of time is not conclusive proof that some degree of impairment does not persist. Physiological evidence from the EEG indicates that abnormalities in the brain wave pattern may last for many months after shock(8), and some patients continue to complain of memory difficulties for several months(3, 9). Whether or not there is actual mental impairment which is not detected by ordinary clinical examinations or which the patient minimizes or fails to recognize is a question that may be answered by psychometric study(11).

There are a number of psychometric studies to be found in the literature(13, 16, 20, 22, 23, 24) but only two of these report follow-up results. Tooth and Blackburn(17), using a modification of the Babcock test, state that 3 out of the 7 patients treated by cardiazol, who showed impairment when tested after shock, continued to show impairment 3 to 7 months after treatment. Wittman and Russell(21) report results on a group tested one year after treatment. While their results indicate a significant improvement in mental efficiency after this interval, it is not possible to determine from these data whether there is residual impairment. Also, a variety of types of disorder and a number of different methods of treatment were involved in that study. For a critical evaluation of the effects of shock on mental efficiency, it would appear desirable that tests be administered a considerable time after the termination of a single type of shock treatment to a fairly large group of patients suffering from psychoses not considered deteriorating in character or involving primary disorders of thinking.

POPULATION AND TECHNIQUE

The patient population used in the present study consisted of 75 persons, 26 men and 49 women. Of these 75, 51 were diagnosed manic-depressive, 22 involutional melancholia, and 2 psychoneurosis with depressive features. Of the 51 manic-depressive cases, 44 were depressed, 5 were hypomanic, and 3 were in the hypomanic phase of the circular type of manic-depressive psychosis. Only patients who cooperated well were included. No patient was suffering from a formal disorder in thinking beyond changes in the rate of mental processes (retardation) or occasional distractibility.

The average age of the group was 41.2 years, with an S.D. of 13.2 and a range of 15-70 years. The patients had been psychotic on the average of 4 to 6 months prior to treatment. The average treatment course was 7.8 electric shocks, the S.D. being 3.3

[1] Read at the 103d annual meeting of The American Psychiatric Association, New York, N. Y., May 19-23, 1947.

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Acknowledgment is made to the following: for assistance in test administration, L. Festinger, G. K. Lumry, A. Kenerson, S. Deri, T. Kendler, E. Hamstra and M. Singer; for copies of the Shipley-Hartford Retreat Scale, Dr. C. C. Burlingame; for assistance in analysis of the data, R. Senf and A. Shoore.

and the range, 1-18. The convulsions were produced by 60-cycle current applied for 0.1 to 0.2 second with a voltage varying from 80 to 150. The electrodes were placed over the temples. The treatments were given at a rate of 2 to 3 per week.

A normal population of 17 individuals matched in age and vocabulary level is included for comparison with our older patients.

The preshock tests were administered 1-3 days before the first shock. The postshock tests were given, on the average, 10.7 days after the last shock, the S.D. being 4.7 and the range 3-30 days. The mean period elapsing before the follow-up test was 189 days, with an S.D. of 99 days and a range of from 36 to 570 days. Since not all of the patients could be tested prior to shock and since some did not return to the hospital for the follow-up examination, the population was divided into three groups: Group A, consisting of 22 patients tested before and after shock treatment; Group B, 21 patients tested after shock and on a follow-up examination; and Group C, consisting of 32 patients who were examined both before and after shock and on a follow-up examination. At the time of the postshock test, 60% of the patients were considered completely recovered from the psychosis and 40% as markedly improved. Of those who returned for the follow-up examination, 85% were completely recovered and 15% had depressive symptoms.

The tests used were the Babcock revision(2) and the Shipley-Hartford(14, 15). These tests were developed originally as measures of "impairment" or "deterioration" of intellectual functions(1, 12, 19). Both are based on the postulate that vocabulary shows very little change with senile or organic deterioration while other intellectual functions show relatively greater changes. In both tests a vocabulary score is used as a basis for estimating the original intellectual level of the patient on functions other than vocabulary, and "deterioration" is measured by the difference between the original level, so determined, and the level of performance at the time of the test.

The Babcock test includes a wide variety of items, 31 in number, which are arranged in 6 groups. The motor tests involve writing and tracing forms. Continuous Work tests speed of performance of simple repetitive tasks. Response to Easy Questions contains such items as giving the days of the week. Repetition involves such tasks as repeating digits forward and similar items. Initial Learning measures ability to reproduce such material as paired associates and paragraphs. Learning deals with the recall of meaningful material after a short delay. In general, mental efficiency as measured by this test primarily involves memory and secondarily attention and speed. A total mental efficiency score is obtained by averaging the scores on these 6 groups of subtests. The efficiency index is derived by subtracting the patient's obtained mental efficiency score from the mental efficiency score expected for a nondeteriorated individual of the same vocabulary level.

The Shipley test consists of a vocabulary test and an abstract thinking test. Impairment is measured by the extent to which the obtained abstract thinking score falls below the expected score for an individual of the same vocabulary level. The efficiency is expressed as a conceptual quotient.

Results

The mean mental efficiency score on the Babcock test for the 54 patients (Group A plus Group C) tested before shock is 13.03 (Table 1). On the postshock test, the mean is 13.35. This slight increase in mental efficiency is not statistically significant. The mean mental efficiency score for the 53 patients who received both postshock and follow-up tests (Group B plus Group C) is 13.25 on the postshock test and 14.58 on the follow-up test. The difference is statistically significant beyond the 1% level of confidence. The trends are similar for the 32 patients who received all 3 tests (Group C). Again the difference from the pre- to the postshock test is not significant, while the difference from the postshock to the follow-up test is highly significant.

The mean efficiency index (Table 1) for the 54 patients who were tested prior to shock is -3.27. The postshock mean for this group is -3.08. The difference is not statistically significant. The mean postshock

score for the 53 patients who were given both postshock and follow-up tests was −2.79 on the postshock test and −1.82 on the follow-up test. This difference is statistically significant. The trends for the 32 patients in the C group alone are similar. The difference between the pre- and postshock scores for this group is not significant, while the difference between the postshock and follow-up scores is highly significant.

up. The trends are similar for Group C, pre- to postshock and postshock to follow-up.

In general, it would appear that the patients as a group showed evidence of impairment in mental efficiency on the preshock and postshock tests, since the mean efficiency indices and conceptual quotients are significantly lower on these tests than on the follow-up tests. Presumably, the impairment on the preshock test is a consequence of psy-

TABLE 1

RESULTS ON BABCOCK TEST

GROUPS A PLUS C ($N = 54$)

	Preshock		Postshock	
	Mean	SD	Mean	SD
Efficiency index	−3.27	2.47	−3.08	2.48
Mental efficiency	13.03	2.93	13.35	2.78
Vocabulary	17.01	2.09	17.48	1.99

Significance of difference between means pre- to postshock: efficiency index, not significant; mental efficiency, not significant; vocabulary, 5%-10% level.

GROUPS B PLUS C ($N = 53$)

	Postshock		Follow-up	
	Mean	SD	Mean	SD
Efficiency index	−2.79	2.29	−1.82	2.21
Mental efficiency	13.25	2.63	14.58	2.54
Vocabulary	16.91	2.06	17.25	1.92

Significance of difference between means postshock to follow-up: all significant beyond the 1% level.

GROUP C ($N = 32$)

	Preshock		Postshock		Follow-up	
	Mean	SD	Mean	SD	Mean	SD
Efficiency index	−3.09	2.35	−3.07	2.28	−2.12	1.90
Mental efficiency	13.05	2.91	13.18	2.56	14.31	2.10
Vocabulary	17.06	2.04	17.22	1.90	17.50	1.85

Significance of difference between means: pre- to post-, none significant; postshock to follow-up: efficiency index, beyond 1% level; mental efficiency, beyond 1%; vocabulary, 5%-10%.

Results on the Shipley test are shown in Table 2. The mean abstract score for the 39 patients tested before and after shock (Group A plus C) is 17.07 on the preshock test and 19.74 on the postshock test. This difference is significant beyond the 1% level. For the 43 postshock and follow-up patients (Group B plus C) the figures are 19.72 and 22.23; this is significant beyond the 1% level. The trends are similar for the 24 patients of Group C. The mean conceptual quotients for Groups A plus C show a probably significant rise from 77 to 80 pre- to postshock. Group B plus C disclose a significant rise from 82 to 86, postshock to follow-

chosis. The impairment existing at the time of the postshock test is not to be attributed to psychosis primarily since 60% of the patients were considered to have recovered from their psychosis and the remaining 40% were markedly improved at this stage. The postshock impairment probably reflects incomplete recovery from the effects of shock. It is doubtful if the changes from test to test can be ascribed to practice since the correlation between the number of days between pre- and postshock tests and change in mental efficiency is −.21 and the correlation between the number of days from postshock to follow-up tests and change in mental

efficiency is −.18. Neither correlation is significant.

The principal question with which we are concerned here is whether electric shock treatment produces any residual impairment of intellectual functioning. The answer to this question, as far as the present study is concerned, is to be found in the performance of the patients on the follow-up tests.

dant evidence to corroborate Gilbert's finding that mental efficiency decreases with age(18). Hence, the patient group, the mean age of which is 41.2 years, should have an efficiency index falling somewhere between 0.0 and −4.8. Their mean efficiency index of −1.82 is significantly lower than that of Gilbert's 20-year-old group and is also significantly higher than that of her 60-year-

TABLE 2

RESULTS ON SHIPLEY-HARTFORD TEST

GROUPS A PLUS C ($N = 39$)

	Preshock		Postshock	
	Mean	SD	Mean	SD
Conceptual quotient	77.08	14.53	80.44	17.09
Abstract score (raw)	17.07	9.26	19.74	10.13
Vocabulary score (raw)	29.10	6.08	30.13	5.30

Significance of difference between means pre- to postshock: conceptual quotient, 5%-10% level; abstract score, beyond 1%; vocabulary, 5%-10%.

GROUPS B PLUS C ($N = 43$)

	Postshock		Follow-up	
	Mean	SD	Mean	SD
Conceptual quotient	81.84	17.23	85.93	14.71
Abstract score (raw)	19.72	10.74	22.23	9.48
Vocabulary score (raw)	29.02	5.45	29.19	5.55

Significance of difference between means postshock to follow-up: conceptual quotient, 1% to 2% level; abstract score, beyond 1%; vocabulary, not significant.

GROUP C ($N = 24$)

	Preshock		Postshock		Follow-up	
	Mean	SD	Mean	SD	Mean	SD
Conceptual quotient	77.58	13.96	82.58	15.97	86.33	14.58
Abstract score (raw)	17.25	9.05	20.33	10.01	22.17	9.54
Vocabulary score (raw)	28.63	5.98	29.38	5.60	29.13	5.89

Significance of difference between means: pre- to post-, conceptual quotient, 2%-5% level; abstract score, beyond 1%; vocabulary, not significant; postshock to follow-up, conceptual quotient, 5% to 10%; abstract score, 2% to 5%; vocabulary, not significant.

Fifty-three patients were given follow-up Babcock tests, with a mean interval of approximately 200 days after the last treatment. The mean efficiency index for this group on the follow-up test was −1.82 with an S.D. of 2.21. Does this efficiency index indicate residual impairment which may be attributed to shock? Norms are not available by which this question may be answered directly. However, in a study by Gilbert(6), the mean efficiency index for 185 normal individuals between 20 and 29 years of age was found to be 0.0, with an S.D. of .97; while for 175 normals between 60 and 69 years old, the mean efficiency index was −4.8, with an S.D. of 1.71. There is abun-

olds. Since the exact shape of the curve for the normal population between 20 and 60 years of age is not known, it is not possible to determine whether the obtained efficiency index of −1.82 is significantly lower than the norm at 41 years of age.

That there is a relationship between mental efficiency and age is borne out by our data. Correlations between age and efficiency index are as follows: Group AC, preshock −.58, postshock −.65; Group BC, postshock, −.53, follow-up −.64. Correlations between age and the conceptual quotient are: Group AC, preshock −.58, postshock −.52; Group BC, postshock −.44, follow-up −.37. All these correlations are

significant beyond the 1% level except the correlation of −.37, which is between the 1%-5% levels.

It seemed desirable, then, to attempt to match the patient population with a normal group of the same age and to determine whether the patients scored significantly lower than the controls. It was possible to match closely 17 of the older patients who had received follow-up tests with normal controls. The control cases were obtained from hospital employees and the community and were matched both as to age and vocabulary level with cases from the patient group. The control group had an age range of 38-68, a median age of 46, a vocabulary range of 18-21, and a median vocabulary of 19. The patient group had an age range of 38-66, a median age of 47, a vocabulary range of 18-21, and a median vocabulary of 19. The mean efficiency index of the control group is −2.0, the median −2.10, the range +1.2 to −5.7. For the patient group the respective figures are: −2.97, −2.9, +1.0 to −7.9. The groups are not significantly different with respect to age or vocabulary, nor are the efficiency indices significantly different. It may be concluded that this group of patients, when tested approximately 6 months after a series of electric shock treatments, shows no evidence of impairment when matched with normal individuals of the same age who have not undergone shock treatment.

This conclusion is supported by the results of the Shipley test. For the 43 patients who were given the Shipley on the follow-up examination, the mean conceptual quotient is 85.93, with an S.D. of 14.71. Using Shipley's norms, which there is reason to believe are too high by 10 to 15 points(5, 10), a conceptual quotient of 85.9 would be considered only "slightly suspicious." When allowance is made for age, or for the strong probability that the norms are too high, the patient group does not appear to show impairment on the Shipley test, some 6 months after shock treatment.

Further light may be thrown on the significance of the scores made by these patients on the follow-up test by an examination of their relative position in the group on the 3 tests. The correlation between the efficiency index scores for Group C on the preshock and follow-up tests is .71; the correlation between these scores on the postshock and the follow-up tests is .76. The preshock conceptual quotients correlate with the follow-up quotients .79; the postshock quotients correlate .80 with the follow-up quotients. These correlations indicate a strong tendency for the patients to maintain the same relative position in the group when tested while mentally ill, shortly after shock, or 6 months following treatment. This points to the possibility that patients who tested low on the follow-up test were also low prior to the development of the mental illness.

Attention is drawn to the stability of the vocabulary scores, preshock, postshock, and on the follow-up (Tables 1 and 2).

From the data obtained, it is also possible to determine the relationship between the number of shocks and the amount of change in test performance. The average number of shocks administered was 7.8, with an S.D. of 3.3 and a range of from 1 to 18 shocks. For the 54 patients who were given pre- and postshock tests, the correlation between number of shocks and improvement in test score is −.32. This correlation, while low, is statistically significant and indicates a slight tendency for patients given more shock to show less improvement on postshock tests. This is consistent with the previous suggestion that the postshock tests reflect a temporary effect of shock. For the 53 patients who were given both postshock and follow-up tests, the correlation between number of shocks and improvement in follow-up scores is .33, which is also statistically significant. On the Shipley test, however, neither of these correlations is statistically significant.

Correlations between age and changes in efficiency index, in mental efficiency, and in conceptual quotient range between +.10 to −.10, indicating that the older patients show relatively as much improvement as do the younger patients, following shock treatment.

Summary and Conclusions

The present study has attempted to determine the effect of electric shock treatment on mental efficiency. The population studied

included 75 patients, suffering from depression, hypomania, or psychoneurosis. The mean age was 41.2 years; the mean number of shocks was 7.8. On Babcock tests administered an average of 11 days after shock, there was a slight but not significant improvement. On the Shipley test, the improvement was probably significant. When the same tests were administered after a follow-up interval averaging 189 days, there was relatively great and statistically significant improvement on both tests. Babcock scores obtained by the patient group about 6 months after shock were not significantly lower than the scores of a normal population of 17 persons of the same age and vocabulary level. It may be concluded that, for the population studied and under the conditions of the present experiment, electric shock treatment does not produce any significant impairment of mental efficiency after an interval of 6 months. No correlations were found between age and improvement from test period to test period. There was some evidence that patients receiving a greater number of shocks were affected more in mental efficiency than those receiving a lesser number of shocks.

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SOMATOTYPES IN RELATION TO DEMENTIA PRÆCOX

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Interest in the relationship between physical constitution and susceptibility to disease goes back as far as Hippocrates and has been lively ever since. The venerable father of medicine described a phthysic and an apoplectic habitus, claiming that elongated physiques were susceptible to tuberculosis while those in which the horizontal dimension predominated tended to have apoplexy(8). Centuries later, a similar dichotomy of peptic ulcer and gall bladder types was proposed by Dr. George Draper and his students(3). In between are to be found a motley procession of medical men, pseudoscientists, phrenologists, physiognomists, and plain charlatans, all espousing theories that different kinds of disease, personality characteristics, or mental powers are associated with various indices of physical form. Beneath all the layers of undeniable quackery, an insistent kernel of truth has kept troubling the repose of those who are interested in human personality and its pathology. Somehow, the make-up of the body did seem to be related to its functioning or malfunctioning. But what kinds of relationships were there, and how could they be conclusively demonstrated?

After centuries of undisciplined surmise and a few years of sterile correlating anthropometric and mental measurements, came the boldest and most striking finding of all. The German psychiatrist Kretschmer reported that he was able to classify the inmates of mental hospitals into three major somatic types and a residual mixed category, the dysplastic(6). His types were the *pyknic,* or stout and compact, the *athletic,* or muscular, and *asthenic* (later changed to leptosomic) or frail and weak. This was nothing particularly new; three-fold classifications of just this sort went back through many slight variations to the French pathologist Rostan (7) and his digestive, muscular, and respiratory and cerebral types. What was startling was that Kretschmer reported a very high concentration—two-thirds—of pyknics among cases diagnosed manic-depressive, while about as large a proportion of the dementia præcox cases fell in the leptosomic and athletic types, in a group of over 4,000 mental patients.

Here seemed to be a finding of great interest and importance, implying to Kretschmer and to many others a relation between physique and personality in the normal as well as abnormal realm. Other workers attempted to duplicate his findings, and a considerable controversy grew up. On the one hand, those who followed Kretschmer's rough and impressionistic method of classifying physiques into general types got results which were consistent with his. On the other hand, the more precise and sceptical workers who attempted the typing of physiques according to rigid criteria came to the conclusion, first, that Kretschmer had no consistent objective system, and second, that their approximations to it yielded no significant relationships with mental disease.

There the matter stood until the anthropologist Sheldon came upon the scene. Well-trained in both medicine and psychology, in the traditions of Kretschmer and of American sophistication in statistics, he succeeded in devising a now widely-accepted technique known as *somatotyping.* He saw that ideal types of the 3 kinds of build did in fact exist, but that most people's bodies were composed of such a mixture and overlapping of the three that a typology was impossible. Instead, he finally set up a system of describing physiques in terms of 3 variables—not of simple dimensions like height and

[1] Formerly, and while this work was being carried on, medical officer, St. Elizabeths Hospital, Washington, D. C.

weight, but of the basic form-qualities that were present in human bodies. The familiar triad reappeared, this time under the names of *endomorphy* for rounded fatness, *mesomorphy* for square muscularity and bigbonedness, and *ectomorphy* for fragile linearity of build. Instead of putting a person into a type according to the component which is most apparent, in the usual way, Sheldon assigns any particular physique a set of three numbers called the *somatotype,* representing the degree to which each component is present. For instance, 1-5-4 indicates a minimum of endomorphy, a stronger than average dose of mesomorphy, and moderate ectomorphy—a lean but powerful body. Though there are infinitely many different bodily forms, only 76 somatotypes were found by Sheldon in 4,000 college men, since he uses a 7-point scale for each component and a person cannot be close to the maximum on more than one.

Unlike Kretschmer's vague indications, Sheldon's system is painstakingly set forth in his book, *The Varieties of Human Physique* (8). Somatotyping can be done by means of a completely objective instrumental method, or, with the expenditure of much less time, independent observers can be trained to a high degree of agreement in classifying photographs in the system of somatotypes.

With the two major objections to Kretschmer's work overcome—its untenable typological approach and the failure of his classification to show inter-observer reliability—it seems high time that his findings be reexamined and duplicated or corrected by means of modern techniques of research. In the book referred to, Sheldon mentions a large study of psychosis in relation to somatotypes, but to date and to our knowledge, he has not published his findings, nor do we know of any other study of somatotypes in relation to psychosis.[2] Meanwhile, the present paper reports the results of an independent attempt to use the somatotyping technique in following up Kretschmer's challenging lead.

Materials and Procedure

Conscious or unconscious selection of cases to fit a hypothesis is a fault which has attended some clinical studies of physique and pathology. Hand in hand with it goes a similar source of spuriously "good" results: knowledge of psychiatric diagnosis when the morphological classification is made. Materials were available to us which allowed for the rigorous exclusion of these sources of bias.

Twelve hundred forty-eight patients in St. Elizabeths Hospital who had successively come to autopsy had been photographed in connection with another study.[3] The photographs were identified by numbers, but otherwise there was no indication on them of psychiatric diagnosis or any other data. They were filed in folders having the same numbers and containing complete case histories, autopsies, and other relevant materials.

Thus we had a large sample of hospital material, uninfluenced by any factor other than death. We took the pictures from the folders, keeping track of them by the serial number only, and did not look up their psychiatric diagnoses until after the somatotyping had been completed. Two important sources of bias were thereby avoided.

Some limitations had to be placed upon the subsample actually used. In the first place, we were interested in somatotyping only cases of dementia præcox, manic-depressive psychosis and general paresis (as a control group). Secondly, since Sheldon had standardized somatotyping on young white males only, non-whites, women, and men over the age of 50 were excluded. The actual age range of subjects was 22 to 50 years, after which time of life the wasting process of old age often sets in; it was realized, of course, that special problems would be raised by the necessity of extending the age range as far beyond 25 as we did. Pa-

[2] As this goes to press, an unpublished paper on the topic by Wittman, Sheldon, and Katz (A Study of the Relationship between Constitutional Variation and Fundamental Psychotic Behavior Reactions) has come to our attention. The results do not bear very directly upon the conclusions of the present study, though they are highly interesting and suggestive.

[3] The authors are greatly indebted to Dr. Walter Freeman, who had taken the original pictures and obtained that material for purposes of his own, in the course of certain anthropometric studies, considerably before Sheldon's work was known at all. He has published his study (4) and it has no particular relationship to ours.

tients who had died of exhaustive diseases such as tuberculosis or cancer were eliminated from consideration, since we thought that it might be too difficult to discount the emaciating effects of the disease. This preliminary selection of cases was made before pictures were removed from the folders.

The number of cases selected by these criteria from the original 1,248 was brought down to 50 cases of dementia præcox, 33 of general paresis, and none of manic-depressive psychosis. It seems at first extraordinary that in a group of 1,248 successive autopsies there should not be a single white male between 25 and 50 years of age who was diagnosed as manic-depressive. This fact can probably be best understood on the assumption that manic-depressive psychosis is a recurrent disease, and that the patients afflicted with it usually die in old age outside an institution. By the same token, mild cases of dementia præcox or paresis in which cure or remission occurred would not be likely to fall into the sample.

Another advantage of the material used is that in practically all cases the diagnosis was firmly established, since by the time the patient came to autopsy he had been in the hospital for quite some years.

With due allowance for the fact that the subjects were dead, the photographs had been taken in close correspondence to the standard procedure described by Sheldon. Each body was photographed held erect against a screen calibrated in 4-inch squares. To support the nude corpse in the upright position, the head was held between calipers. A professional photographer took the pictures 3 on a plate: front, back, and side views.

It was in order to avoid still another criticism which has been made of Kretschmer's work that we decided to include a control group without psychogenic psychosis. Of course, Sheldon's normal college series could be used, but it seemed advantageous to choose a group of cases all of which had been subjected to the same hospital environment, photographed and somatotyped in the same way by the same persons. Our original intention was to use cerebral arteriosclerotics as controls, but it became quickly apparent that this group was composed almost entirely of persons over the age of 50, and therefore had to be discarded. Paretics were our next choice because it seemed that there was probably no very important selective factor differentiating from the general population people who contract syphilis. We were aware of the existence of a little-accepted hypothesis that certain constitutions may be particularly inclined to develop neural complications of syphilis.[4] We did not believe, however, that this consideration was weighty enough to prevent our using the paretic group for a control.

The two authors somatotyped each picture separately and independently of each other, recorded the somatotype with the case number, and then compared results. When there were differences, we went back to the picture and discussed the evidence in detail, finally agreeing on a somatotype of each case. Inter-observer reliability for the 3 components ranged from .69 to .77, while the correlations of the observers with the consensus ranged from .88 to .93 (Table 1).

TABLE 1

RELIABILITY OF SOMATOTYPE RATINGS

	Endo-morphy	Meso-morphy	Ecto-morphy
Author 1 *vs.* Author 2..	$r = .75$	$r = .69$	$r = .77$
Author 1 *vs.* Consensus..	$r = .88$	$r = .89$	$r = .89$
Author 2 *vs.* Consensus..	$r = .90$	$r = .88$	$r = .93$

$N = 83$; when $N = 80$, r's of .29 or larger would be obtained less than once in a hundred random samples from a population in which the true $r = .00$.

Since the somatotype is a pattern, in which the relative dominance of the three components is at least as important as their magnitudes, correlations do not furnish an altogether satisfactory measure of reliability. On the other hand, there are no norms against which to judge the excellence of other reliability scores. It may still be worth recording that in 13% of the cases there was disagreement on the major dominance, such as the difference between a 3-4-5 and a 3-5-4; in 42% of the cases there was agreement on the major dominance but disagreement on the minor, as for instance 3-4-5 against 4-3-5; while in 45% of the cases both observers agreed on both major and minor dominance of components.

[4] The interesting psychosomatic study of Kenyon, Rapaport, and Lozoff (5) which lends more weight to a theory of psychogenesis and personality diathesis in paresis may indirectly support this point of view.

The somatotyping was done by what Sheldon calls the method of anthroposcopy, which is visual inspection of the photograph while the many indicators of each component are kept in mind. An effort was made to check our consensus against the objective anthropometric technique described by Sheldon, making use of his tables. A serious difficulty prevented us from completing this check. The measurements which Sheldon describes could be made easily enough from the photographs, but they could not all be used in connection with Sheldon's tables. His norms are based on the measurements of his 4,000 unselected college men, all of whom are between 18 and 25 years of age. Now, many of the diameters measured, such as the breadth of the head above the ears, do not change with age, since they are almost entirely determined by the bony structure. Others, such as the depth of the abdomen, do change greatly in later years, as many of us know to our sorrow. Sheldon claims that this kind of development does not change the somatotype, but obviously if the somatotype is to remain constant, the tables of norms for it must be recomputed anew for each new age range. Results from a partial application of the technique, using only measurements which change least with age, did correspond rather closely to the agreed somatotype by the inspectional method, but the full list of measurements is necessary to make accurate determinations of somatotypes for the body as a whole.

Results

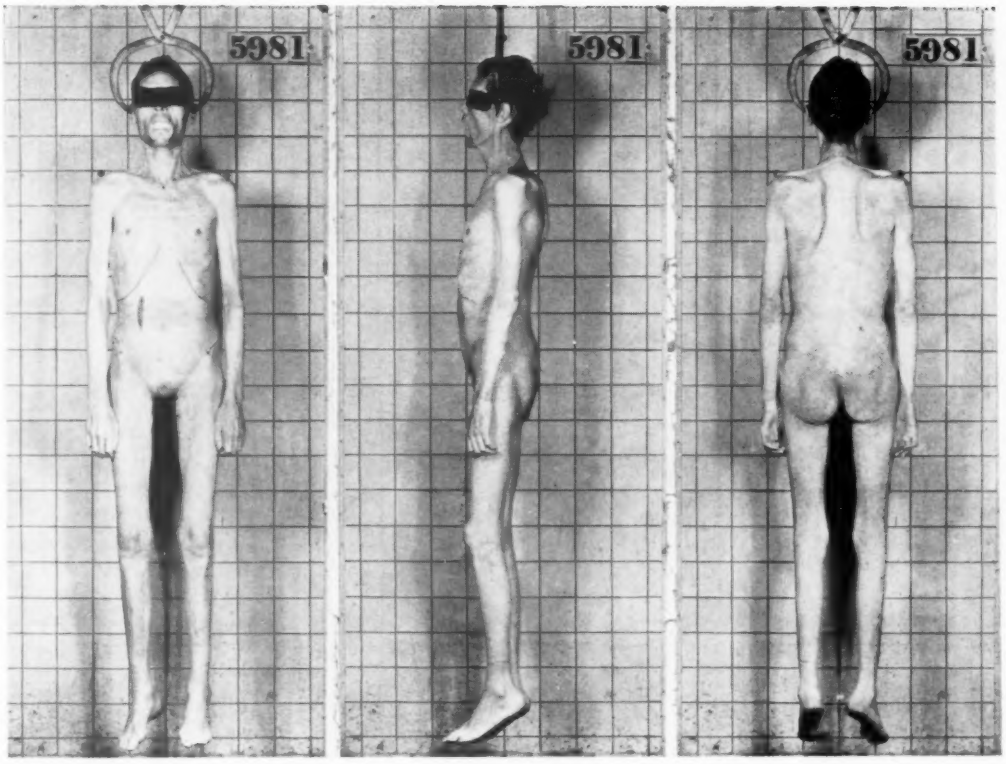
The distribution of the somatotypes in each clinical group is compared with Sheldon's figures on college men as a standard in Table 2. It may be seen that while the distributions for dementia præcox and paresis are rather similar, both differ considerably from the college group. The simplest way to summarize the differences is to report the "mean somatotype"—that is, the mean value of each component—for each group. The DPs (dementia præcox cases) average on the three components 2.2 (endomorphy)—3.2 (mesomorphy)—4.5 (ectomorphy); the paretics average 2.2—3.1—4.7; while the college men average 3.2—3.8—3.5.

There is no significant difference between the mean ratings on any component of the DPs and the paretics, but every comparison between these groups and Sheldon's college men yields a significant difference. Fisher's *t* for the three comparisons between the psychotics is entirely insignificant, being only .1, .3 and .6. When the DPs and paretics are compared with the college men on the 3 components, however, critical ratios are quite significant, ranging from 3.2 to 5.2. Both experimental and control subjects are thus, on the average, significantly more elongated and thin, less soft and fat, and less square

TABLE 2

Distributions of Somatotypes Among Cases of Dementia Præcox and Paresis

Somatotype	Incidence in cases of: Dementia præcox N	Dementia præcox %	Paresis N	Paresis %	Sheldon's norms (college men) %
1-1-7	2	4	1	3	.5
1-2-6	8	16	4	12	.7
1-2-7	2	4	2	6	.5
1-3-6	2	4	3	9	.2
2-1-7	1	2	0	0	.1
2-2-5	5	10	2	6	2.0
2-2-6	0	0	1	3	2.5
2-3-5	5	10	6	19	4.5
2-3-6	1	2	0	0	.5
2-4-4	3	6	3	9	4.2
2-4-5	2	4	1	3	1.4
2-5-4	0	0	1	3	1.6
2-6-3	1	2	0	0	.6
2-7-1	1	2	0	0	.3
3-3-4	1	2	1	3	5.0
3-4-3	2	4	0	0	4.7
3-4-4	2	4	0	0	5.8
3-4-5	1	2	0	0	.8
3-5-2	2	4	0	0	2.3
3-5-4	1	2	0	0	1.3
3-6-1	1	2	0	0	.6
3-6-2	0	0	1	3	.9
4-3-3	0	0	2	6	2.4
4-3-4	1	2	0	0	3.5
4-4-2	0	0	1	3	2.9
4-4-3	2	4	1	3	5.6
4-5-2	1	2	1	3	2.3
4-5-3	0	0	1	3	1.0
4-6-1	1	2	0	0	.1
5-3-2	1	2	0	0	1.8
5-3-3	0	0	1	3	2.4
6-2-2	1	2	0	0	.5
All others	0	0	0	0	35.5
	50	100	33	100	100.0



SAMPLE SOMATOTYPE PICTURE: A 40-YEAR OLD PATIENT, WHO HAD BEEN HOSPITALIZED NEARLY 17 YEARS FOR DEMENTIA PRÆCOX. SOMATOTYPE: 2-2-5.

and muscular than the supposedly normal sample somatotyped by Sheldon.

As we have said, part of the value of the somatotyping method is that it considers the total configuration of the physique. Much of the possibly fine discrimination between kinds of builds is lost if means of the components alone are used. Unfortunately, the number of cases is too small to permit comparison of the obtained distribution of somatotypes with expectations on the basis of Sheldon's figures. Some grouping of related somatotypes is necessary so that the Chi-square test of the significance of difference between 2 distributions may be used.

Sheldon suggests that for purposes of research it may be best to put together similar somatotypes, and he gives a suggested grouping which reduces the number of categories from 76 to 19. This was still too many for our purposes, but working from it and combining related groupings until the theoretical frequencies were large enough, we ended up with 5 general classes of somatotypes, grouped on the basis of the relative dominance of the components (Table 3).

It is not surprising to find that the Chi-square test confirms the critical ratio, and that the somatotype distributions of the DPs and paretics are very significantly different from the expected frequencies derived from Sheldon's norms. The added advantage of Chi-square is that it enables us to fractionate the results, and see which categories contribute significantly to the total measure of difference.

To start with, consider the *first class* of *somatotypes*. Here are all the physiques in which endomorphy is dominant, plus the endomorphic mesomorphs—that is, those in which there is a secondary dominance of endomorphy in predominantly mesomorphic bodies, and the endomorph-mesomorphs, who are equally endowed with the first two components at the expense of the third. This class contains all the large bulky and blocky builds in addition to the frankly fat ones, and probably comes close to what Kretschmer would have called the pyknics. They are underrepresented in the dementia præcox sample, but not significantly so; there are practically as many of them among the paretics as would be expected theoretically.

The *second class* is composed of all the rest of the physiques in which mesomorphy is dominant, including those in which there

TABLE 3

COMPARISON BETWEEN DISTRIBUTIONS OF SOMATOTYPES. PSYCHOTICS VS. SHELDON'S NORMS

Somatotype group	Obtained *N*	Theoretical *N*	Chi-square
DEMENTIA PRÆCOX			
All endomorphs, endomorphic mesomorphs, and endomorph-mesomorphs	8	13.4	2.2
Predominant mesomorphs and ectomorphic mesomorphs	4	11.0	4.5[a]
Mesomorphic ectomorphs and mesomorph-ectomorphs	25	6.7	50.0[b]
Predominant ectomorphs, endomorphic ectomorphs, and ectomorph-endomorphs	8	9.4	.2
Balanced physiques	5	9.5	2.1
Totals	50	50.0	$\chi^2 = 59.0$[c]
PARESIS			
All endomorphs, endomorphic mesomorphs, and endomorph-mesomorphs	7	8.9	.4
Predominant mesomorphs and ectomorphic mesomorphs	1	7.3	5.4[a]
Mesomorphic ectomorphs and mesomorph-ectomorphs	19	4.4	48.4[b]
Predominant ectomorphs, endomorphic ectomorphs, and ectomorph-endomorphs	5	6.2	.2
Balanced physiques	1	6.2	4.4[a]
Totals	33	33.0	$\chi^2 = 58.8$[c]

[a] When Chi-square = 3.8, with 1 degree of freedom, $p = .05$.
[b] When Chi-square = 6.6, with 1 degree of freedom, $p = .01$.
[c] When Chi-square = 23.5 with 4 degrees of freedom, $p = .0001$.

is no secondary dominance, and the mesomorphs in which ectomorphy is stronger than endomorphy. These are all large-boned, heavily muscled builds, though some of them, where ectomorphy has secondary dominance, are tough and wiry rather than square and solid. They are significantly underrepresented in both the hospital groups.

In the *third class* are the mesomorphic ectomorphs; that is, only those physiques in which the third component of lean angularity is accompanied by a subdominance of muscular and bony strength instead of any softness or tendency to fat. This kind of build is strikingly and very significantly prominent among both the DPs and the paretics, well over half of the latter group being mesomorphic ectomorphs instead of the "normal" expectation of about one in seven. The mesomorph-ectomorphs, who have equally strong second and third components, are included here too.

All other ectomorphs, including those having endomorphy as a secondary dominance, make up the *fourth class*. People who are simply slim, sharp-featured and narrow-chested come under this classification, as well as a few who have also some weak pudginess on thin slender bones, without any muscular solidity. This category makes the smallest contribution to Chi-square of any; both DPs and paretics have almost exactly the expected numbers of these ectomorphic cases.

Finally, the *fifth class* comprises the balanced physiques in which no one component is dominant. Fewer of them are found in both the clinical groups than would be expected from the null hypothesis, but the difference is significant only in the case of the paretics.

The Chi-square test thus reveals that the differences in mean ratings of the components are not due to a generally increased ectomorphy among the psychotics and a uniform deficiency of the other two components. *It seems rather that the differences are due almost entirely to the extremely high incidence of mesomorphic ectomorphs in the two hospital groups.* So far as endomorphy is concerned, nearly as many cases of endomorphic physiques are found as would be expected from the null hypothesis, but that component does not appear to be highly developed in the psychotic patients, even when it is dominant in their physiques.

TABLE 4

COMPARISON BETWEEN DISTRIBUTIONS OF SOMATOTYPES, EXPERIMENTAL AND CONTROL GROUPS

Somatotype group	Obtained *N*		Theoretical *N*	
	DP	Paresis	DP	Paresis
All endomorphs, endomorphic mesomorphs, and endomorph-mesomorphs	8	7	9.1	5.9
Predominant mesomorphs, ectomorphic mesomorphs, and balanced physiques	9	2	6.6	4.4
Mesomorphic ectomorphs and mesomorph-ectomorphs	25	19	26.5	17.5
Predominant ectomorphs, endomorphic ectomorphs, and ectomorph-endomorphs	8	5	7.8	5.2
Totals	50	33	50.0	33.0
			Chi-square = 2.6 *	

* When Chi-square = 2.4, with 3 degrees of freedom, $p = .50$.

Table 4 presents a similar comparison between the two psychotic groups. Classes two and five were combined since theoretical frequencies would have been too small otherwise for valid application of the Chi-square test. There is no indication of a significant difference in the distributions of somatotypes, any more than there is between the mean values of any one component, in the 2 groups.

DISCUSSION AND INTERPRETATION

We are left with 2 questions to be answered: First, why was there no difference between the somatotype distributions of our experimental and control hospital groups? Second, why were there significant differences between both of our groups and Sheldon's? Several possible answers suggest themselves for both questions.

A. Why similar somatotypes among DPs

and paretics? Two lines of argument may be followed:

1. *The two groups were actually different in somatotype, but:*

a. *We erred in our judgments of their somatotypes enough to disguise the differences.* Against this contention it may be argued that we had had instruction from Sheldon in the anthroposcopic technique, in addition to having most if not all of the experiential requirements he lays down for somatotypers(8). During the course of the somatotyping, we referred often to his descriptions of the somatotypes and to sample photographs of each of them.

b. *Though the two groups had had different physiques before entry to the hospital, subjection to the same diet, hospital régime, and lack of exercise had either actually changed their somatotypes or changed their external appearance toward similarity to such an extent that we called them the same.* It is true that there was considerable paralysis in the paretic group, and that many of the DPs were so inert as to be effectively paralyzed; in both groups there were a few patients who had had to be tube-fed. Yet influences of this kind would not necessarily make men into mesomorphic ectomorphs. When an endomorphic person takes no exercise, he gets more rotund if reasonably well nourished, not less; but these samples are *not* alike in containing many people in whom accretions of fat have disguised underlying differences.

In favor of this line of argument it may be admitted that many of the patients did in fact seem to be emaciated; we tried to allow for it but may not have succeeded. It is easier to see the traces of mesomorphy in an emaciate than those of endomorphy, which might help to account for the large number of mesomorphic ectomorphs.

2. *Both groups are actually samples drawn from the same population.*

a. *It might be maintained that physique has no relation to mental disease, so no two groups of patients should be expected to be any different from each other or from the general population.* This line of argument seems effectively negated by the considerable deviation of both groups from Sheldon's norms.

b. *It could be maintained that the clinical diagnoses were so poor that both groups are equivalent samples from a homogeneous hospital population, which may be different from people in general.* Since there was direct autopsy evidence of neuro-syphilis in all cases diagnosed as paresis, this line of argument would have to maintain that the syphilis merely helped to precipitate psychosis in the control group, who were as much constitutionally predisposed to it as the experimental group. In effect, this would be saying that the results mean only that psychotics who have had syphilis have bodily builds similar to those in a mixed nonsyphilitic psychotic group.

The hypothesis has been advanced that a definite constitutional type tends to acquire cerebral complications of syphilis. These data might be interpreted as supporting the view that mesomorphic endomorphs are constitutionally susceptible to paresis if they are infected with lues.

c. *One of the authors holds that dementia præcox is not a disease entity but a reaction type to a great variety of etiological factors, ranging from complete psychogenicity to complete organicity.* He has also advanced the idea(1, 2) that one can consider certain cases of paresis which can practically not be differentiated *clinically* from a case of schizophrenia, to be a possible demonstration of a schizophrenic syndrome with completely organic etiology.

The idea occurred, therefore, that maybe the clinical reaction type might be a matter of constitution. That is, it was thought that paretics showing an essentially schizophrenic clinical picture might be people with a bodily constitution like schizophrenics.

Thus, we investigated those 18 paretics who outstandingly contributed to the ectomorphy of the sample. We felt that they might possibly show a more schizophrenic-like clinical-psychiatric picture than the other paretics. We were disappointed, however: there was practically no schizophrenic syndrome among the 18 paretics who were particularly ectomorphic. If anything, there were less such cases in that sample than we should have expected from experience. We do not feel that this problem is entirely settled in our minds, however, since it turned

out that a surprisingly large percentage of this group came to the hospital in a rather advanced stage of paresis with the deteriorative features outstanding. We believe that schizophrenia-like features occur more often with the early stages of the disease.

B. Why does our internally uniform hospital sample differ from Sheldon's general population?

1. *Our sample is merely an unusual normal one.* But: our low *P*-values make this very unlikely.

2. *Mesomorphic ectomorphs merely die off faster, are more susceptible to disease, so are likely to get into a sample of corpses.* But: Sheldon says the opposite, though he offers no proof beyond clinical experience.

3. *Sheldon's sample is not as normal as ours.* College men are economically and socially more privileged than others, so are better nourished and may look more endo- and mesomorphic. But: Sheldon claims somatotyping is not affected by degree of nourishment. Good athletes get scholarships and other encouragement to go to college; therefore the great mesomorphy of the standardization group. But: scrawny intellectuals get scholarships too! Sheldon thinks his college men have less endomorphy than the general population, not more, as would have to be the case if our sample were more representative than his.

Also a biasing factor, possibly, in his sample: if being somatotyped were at all voluntary, people who deviate from prevalent standards of manly beauty, the very ectomorphic (and also very endomorphic), would tend to stay away, giving too much mesomorphy.

The aggregate of such possible biases in Sheldon's normative group would not seem to be enough to account for the differences obtained.

4. *Judgmental bias.* Perhaps there is no real difference between Sheldon's group and ours, and we merely did not somatotype the way he did. This criticism cannot easily be refuted; we can only remind the reader of our training by Sheldon, and our efforts to follow his system as closely as possible.

5. *Unreliability of our judgments.* Chance differences between groups would still be very unlikely even if our inter-observer reliability fell as low as .60.

Of all these objections to the apparent significance of our findings, we felt that the most cogent was the argument that subjection to the hospital environment, conative or muscular paralysis, and physical illness leading to death may have had similar effects on many patients in both groups. We had tried to distinguish between original ectomorphy and emaciation, but we did feel that it was plausible that common influences of a nonconstitutional nature could have made all the subjects look more alike and look more ectomorphic.

Consequently, we decided to make one more attempt at a control: a number of patients who had been subjected to the same hospital environment and who also had died. If the preceding hypotheses were correct, these nonschizophrenic, nonparetic cases should show the same effects. We decided, then, to somatotype a sample of all the remaining cases, still keeping the same criteria of age, ethnic group, sex, and absence of wasting disease. It was surprising to find that only 15 such cases turned up. Since this was such a small group, and since there were an extra 10 who met all the standards except that of age, being from 51 to 60, we decided to include them on the chance that no bias would be introduced.

TABLE 5

RELIABILITY OF SOMATOTYPE RATINGS, FINAL GROUP

	Endomorphy	Mesomorphy	Ectomorphy
Author 1 *vs.* Author 2..	$r = .85$	$r = .70$	$r = .82$
Author 1 *vs.* Consensus..	$r = .96$	$r = .86$	$r = .90$
Author 2 *vs.* Consensus..	$r = .95$	$r = .85$	$r = .95$

$N = 25$; r's of .51 would be obtained less than once in a hundred samples of this size from a population in which the true $r = .00$.

The procedure of somatotyping was slightly different in this second series of judgments. We were by now considerably separated in space, so we rated the pictures separately and mailed each other our ratings when both had finished. After a minimum of consultation by mail, an agreement was reached on each case—usually by taking the mean of the 2 independent ratings. As can be seen by a comparison of Tables 1 and 5, the reliability coefficients compare well with those obtained under presumably more favorable conditions. It was necessary next to de-

cide whether the additional older 10 subjects could be included in the control group. The mean ratings on the 3 components for the 2 subgroups were as follows:

Age 20-50....2.9—3.3—4.1
Age 51-60....3.1—2.4—4.2

Although even the largest of the three differences, that in the second component, is insignificant ($t=1.2$; $P>.20$), and though ectomorphy is about equally important in both, the older men are on the average more endomorphic than mesomorphic, reversing the relationship found in all other groups in the present study. Furthermore, if the older patients are included in the control group, there is a significant difference between its mean endomorphic rating and those of the DPs and paretics ($t=2.7$, 3.4 respectively; in both cases, $P<.01$), while, if they are excluded, all differences between controls and the other 2 groups are insignificant ($t=1.38$ to .3). It seemed best, therefore, to discard the data on the older patients, even though when they were included and the somatotypes were clustered into 5 large categories, as before, the Chi-square test showed that the control group still did not differ from either of the others. Whether or not the older patients were included, the same familiar pattern appeared: a great excess of mesomorphic ectomorphs and a correspondingly large deficit of strongly mesomorphic builds, with about the expected number of other kinds. With the small number of cases in the reduced control group, the Chi-square test can be used only if all ectomorphic physiques are pitted against all others. The obtained distribution differs from theoretical expectation on the basis of Sheldon's norms, though the Chi-square of 2.86 only approaches significance (P between .10 and .05; Yates' correction used). This 2 x 2 comparison of control with DPs and paretics yields insignificant Chi-squares, indicating similar distributions (DP: Chi-square=.03, P between .80 and .90; paretics: Chi-square=1.13, P between .20 and .30).

TABLE 6

MEAN SOMATOTYPE RATINGS OF ALL PATIENTS BY LENGTH OF STAY IN HOSPITAL AND AGE

Subgroups of total sample	Mean rating on: Endomorphy	Mesomorphy	Ectomorphy	Number of cases
LENGTH OF STAY IN HOSPITAL				
6 months or less	1.91	3.15	4.98	23
More than 6 months, to 2 years	2.72	3.84	3.59	16
More than 2 years to 5 years	3.27	3.00	4.73	22
More than 5 years	2.30	2.97	4.62	33
AGE AT TIME OF DEATH				
22 to 30 years	2.17	2.83	5.17	15
31 to 40 years	2.21	3.27	4.50	35
41 to 50 years	2.35	3.21	4.40	44
51 to 60 years	3.10	2.40	4.20	10
Total sample (excluding age 51-60)	2.27	3.17	4.56	94

Such a long parade of insignificant differences forces us to the conclusion that, in the population studied, similar somatotypes were found in all nosological groups. The explanation is still obscure, however; is the lack of difference due to the lack of any relation between somatotype and mental disease in spite of the fact that all groups differ significantly from Sheldon's "normal" population, or is it due to a tendency of the hospital environment and prolonged illness of any kind to make homogeneous originally diverse physiques by the effects of little exercise and poor nutrition?

An analysis of the data by length of hospitalization enables us to rule out the last hypothesis. Table 6 presents the means for each component in 4 groups of patients: those who had been in the hospital 6 months or less, more than 6 months to 2 years, more

than 2 years to 5 years, and more than 5 years. In the last category there are many patients whose stay in the hospital exceeded 20 years. As can be seen from an inspection of the means, there are no consistent trends, though considerable differences appear. Table 7 presents analyses of the variance of the ratings on each component and indicates that these differences between length-of-hospitalization groups approach significance for endomorphy and mesomorphy and are significant near the one percent level for ectomorphy. Interestingly enough, it is the group who were in the hospital the *shortest* time who had the least amount of fattiness, and who were the most gaunt, elongated, and fragile! It may be that they were more or less moribund, in a very poor physical condition, when they arrived, but the fact that those who died after having been in the hospital more than 6 months but no longer than 2 years closely approach the means of Sheldon's group is very difficult to explain. Unfortunately, the case folders are no longer available to us, so we cannot look for the further data about these people that might explain such curious results.

TABLE 7

ANALYSIS OF VARIANCE OF SOMATOTYPE RATINGS GROUPED BY LENGTH OF HOSPITALIZATION

Source of variance		Sum of squares	D.f.	Mean square	F	P*
Endomorphy	Between groups	10.87	3	3.29	2.29	$> .05$
	Within groups	143.57	100	1.44		
	Total	154.44	103			
Mesomorphy	Between groups	9.46	3	3.15	2.35	$> .05$
	Within groups	134.08	100	1.34		
	Total	143.54	103			
Ectomorphy	Between groups	28.22	3	9.41	3.87	$< .05$
	Within groups	242.97	100	2.43		$> .01$
	Total	271.19	103			

* For 3×100 d.f., an F of 2.70 is significant at the .05 level, an F of 3.98 at the .01 level.

The unexpected deviance of the 10 patients who were over 50 brought up the question of the effect of age on somatotype. Table 6 shows a steady increase in endomorphy with advancing age, and just as consistent a decline in ectomorphy, while mesomorphy first increases a little after 30, then drops sharply in the sixth decade. The correlation between age and endomorphy is only +.20, that between age and ectomorphy is −.20 ($N=104$, $P=.05$), while age and mesomorphy are unrelated ($r=.005$). Still, age seems to be more clearly related to somatotype than does any other variable used in the present study.

Critics of somatotyping often object that physiques change considerably as people get older; the lean youth becomes more and more like his paunchy father as his hairs turn gray and drop out. Sheldon, while accepting the latter observation, has claimed that the basic pattern does not change, but only becomes more clear-cut; in his book (8) he describes the kinds of changes that maturity brings about in different kinds of somatotypes, giving a number of illustrations. We read these sections of the book with particular care and tried to apply the principles laid down. Perhaps we merely failed to see the underlying identity of superficially different physiques. These data cannot be said to disprove Sheldon's claim that the somatotype is stable, therefore, even though they do cast some shadow of doubt upon it. Just as plausible an explanation would be that longevity instead of age is the true correlate of endomorphy and ectomorphy, at least in the present ranges. Excessively fat persons are known to have poor life expectancies, but there was no patient in the population studied who was fat enough to have put much of a burden on his heart.

Conclusions

The empirical finding of the present study can be stated simply enough: mental patients who have died of other causes than exhaustive diseases include in their number a strikingly large proportion of persons with long, lean, wiry, not merely ectomorphic but to some extent mesomorphic physiques, and a conspicuous lack of those with big-boned, heavily muscled builds. They form a relatively homogeneous group, differing not at all by the nature of their mental illnesses, whether functional or organic in origin: but those who were older at the time of their deaths tend to be bulkier, more rounded, and less thin and fragile than those who died at early ages.

To explain these facts is still difficult. Perhaps the patients really differed from an unselected population in these respects before the onset of their illnesses, in which case, one would be led to a hypothesis of a constitutional make-up with nonspecific vulnerability to psychosis. Very probably, the bodily build is not the result of the mental illness, directly or indirectly; if it were, one would expect the effects to show more sharply in cases where the disorder was of long duration. It does not seem plausible that faulty technique on the part of the investigators could have produced such consistent results if readily apparent differences between nosological groups did exist. Possibly mesomorphy is less apparent in a person who is suspended in mid-air instead of standing up, and dead—without muscular tonus—instead of alive; other bias introduced by the fact of death seems less probable, since there were a number of suicides and accidental deaths and none after an exhaustive, lingering illness.

It is possible also that some of the differences between our subjects and Sheldon's are attributable to lack of representativeness in his sample as well as in ours. If it is also true that somatotypes change with age, another reason for not using Sheldon's norms as a standard might be found, though such a hypothesis would make the results even harder to explain. The group which is most comparable in age with the 4,000 college students is the very one which is least like them, while advancing age puts enough flesh on these skinny patients to make them appear (perhaps speciously) more like the standardization group. It is credible that American men as a group are not as athletic in their physiques as young college men, but it is very hard to believe that they are as scrawny as the majority of our research population. The general finding of a specific excess and deficit of just two kinds of builds, making up a total of only 35.5% of Sheldon's group, is thus hard to explain away on the basis of sampling error. A possibility which does still remain is that one of these groups—the mesomorphic ectomorphs—is simply less viable, while the other—the predominant mesomorphs and ectomorphic mesomorphs—has greater vitality. It would be easy to test this hypothesis by somatotyping a similar group of patients dying successively in a general hospital, using the same sampling controls as the ones we employed. If significant differences between such a group and ours were still found, it would be possible to say with some confidence that the peculiarities of somatotype were specific to mental illness.

In *The Varieties of Human Physique*, Sheldon has already hedged a little against the possibility of largely negative findings such as the ones presented here, by stating that certain kinds of dysplasia are much more closely related to dementia præcox than are particular somatotypes. The analysis of dysplasia, or disharmony between various regions of the body, requires an additional five sets of judgments for each picture; we decided to forego it. The possibility still remains, then, that more refined and exhaustive somatotyping studies (and we may add, much more time-consuming ones), might bear fruit of positive results if oriented toward the analysis of dysplasia.

The difficulties which have plagued this study point clearly to one conclusion: the mere adoption of the technique of somatotyping opens up no royal road to the relationship between physique and psychosis. As it presently stands, the system is not well adapted to application to the very types of research problems to which it seems to have so much to contribute. To have studied with the originator of the technique and to have tried conscientiously to follow the book is not enough to enable one to apply it to subjects who differ appreciably from those

Sheldon used and end up confident that the results are not a function of errors in method. Until normative studies have been done upon satisfactory samples of older men, and of women, somatotyping studies will perforce be limited to populations of white college men or others who are similar in sex, ethnic group, and age.

Within these limitations, it is still possible to design a study which can result in conclusive findings where we have tried to pioneer. The research population should be a complete or a random sample of hospitalized patients, all physically healthy white males between 18 and 25 years old. They should be somatotyped independently by two or more observers who have no knowledge of the patients or their diagnoses, preferably using the anthropometric technique or at least validating anthroposcopic judgments by measurements and reference to the tables in Sheldon's book in a considerable subsample. Groups should be equated for length of hospitalization and for duration of illness. Diagnoses should be clearly and of course independently established, if possible by the use of psychological tests as well as clinical examination. Finally, the data should be analyzed from the standpoint of mean ratings, groups of somatotypes, and kinds and degree of dysplasia. Such a study, which would not be prohibitively difficult to carry out, would furnish the conclusive data which are so badly needed on the relationship between mental disease and physique.

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THE FREQUENCY OF SYPHILITIC PRIMARY OPTIC ATROPHY IN DEMENTIA PARALYTICA[1]

A Clinico-Anatomic Study

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Since Alzheimer(1) in 1904 made the statement that in general paralysis there is rarely an involvement of the optic nerves, this viewpoint has been accepted by many subsequent authors(2, 3). Wilbrand and Saenger(4), a few years later, made the additional comment that optic atrophy occurs only in general paralytics who show involvement of the posterior columns of the spinal cord.

Among the first to make detailed studies of the fundus of patients with dementia paralytica were Raviart and Caudron(5). They found the number of pale optic disks increasing as the disease advanced. In a group of 44 general paralytic patients there was complete bilateral optic atrophy in 1 patient, and in 12 others the optic disks revealed "paling." Billod(6) in 400 general paralytics observed blindness in 3 instances. On the other hand, Léri(7), the most outstanding earlier student of this subject, found 3 complete optic atrophies in 60 general paralytics. He also emphasized that slighter changes of the optic pathways were by no means rare in dementia paralytica. Jakob (8) expressed the view that the estimate of optic atrophies among general paralytics, as given by Gudden (4.9%), Kraepelin (4 to 9%) and Mendel (12%), was too high. Rea(9) in his book on Neuro-Ophthalmology cites the prevalence of optic atrophy among general paralytics as approximately 8%, in contrast to tabes dorsalis where optic atrophy occurs in 20%. He adds that it is in the terminal stage of general paralysis that complete atrophy of the optic nerves is liable to take place. Yet it has been seen to appear at an early period and has preceded mental symptoms by several years.

According to these authors the incidence of blindness in dementia paralytica due to syphilitic optic atrophy ranges between 1 and 10%.

In my own clinical material, comprising 1,000 cases of general paralysis, complete or near complete optic atrophy was present in 2%. In addition, unilateral optic atrophy was observed in 4 instances.

Incipient and intermediate stages of syphilitic optic atrophy in general paralytics are always missed, unless the patient is able to cooperate in a perimetric examination. Beginning syphilitic optic atrophy is manifested by constriction of the visual fields, yet the optic discs may still be within the range of normal. It is not generally known, however, that even complete loss of vision is not infrequently overlooked because the patient will not tell of its presence or he is not capable to note its existence.

Denial of blindness by patients with cerebral disease was first described by Anton (10). In Anton's cases and in those reported by others(11, 12), the blindness was usually due to bilateral hemianopsia as the result of lesions in both occipital lobes and optic radiations. Pötzl(13) directed attention to the fact that unawareness of blindness or the denial of own blindness was frequent in general paralytics with optic atrophy. If one questions such a patient as to his eyesight, he will answer: "My eyes are perfect," or "I have the best eyes in the world." When confronted with the fact that he is blind, he will say: "I can see perfectly, if I have my glasses," or give some other quite plausible excuse. Pötzl(13) believed that Anton's syndrome in blind general paralytics is independent of the disturbance of memory and of other abnormal mental manifestations such as euphoria. This is contrary

[1] Read at the 103d annual meeting of The American Psychiatric Association, New York, N. Y., May 19-23, 1947.

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to my observations. Denial of blindness was observed only in acutely psychotic paralytics and in those with severe defects in memory and a tendency to confabulation. This is illustrated by the following instance. A euphoric patient denied his blindness in the initial stage of dementia paralytica but admitted it freely after he had recovered from the mental symptoms following a successful course of malaria therapy.

Material and Result of Anatomic Study

Since incipient optic atrophy in general paralysis is difficult to ascertain clinically, an anatomic study of the optic pathways of 40 general paralytics was made. There were 27 cases of general paralysis and 13 instances of taboparalysis. A diagnosis of taboparalysis was made, when on histologic examination the slightest degeneration was observed in the posterior columns of the spinal cord.

TABLE 1

Forty Patients, Including General Paralysis and Taboparalysis

	No. of cases
Complete optic atrophy	8
Unilateral optic atrophy	1
4 plus marginal degeneration of the optic nerves.	2
3 plus marginal degeneration of the optic nerves.	1
2 plus marginal degeneration of the optic nerves.	5
1 plus marginal degeneration of the optic nerves.	11
No marginal degeneration of the optic nerves..	12
Total	40

TABLE 2

Twenty-Seven Patients With General Paralysis

	No. of cases
Complete optic atrophy	4
Unilateral optic atrophy	1
4 plus marginal degeneration of the optic nerves.	2
3 plus marginal degeneration of the optic nerves.	1
2 plus marginal degeneration of the optic nerves.	4
1 plus marginal degeneration of the optic nerves.	7
No marginal degeneration of the optic nerves..	8
Total	27

The optic pathways were fixed in formaldehyde, embedded in celloidin and cut serially. Alternating sections were stained with toluidin blue and with Weil's myelin sheath method(14). The use of these two stains permitted the simultaneous observation of both the inflammatory and the degenerative phase of syphilitic optic atrophy.

In a previous study(15) from this laboratory it was reported that in the majority of instances the inflammatory-degenerative process of syphilitic primary optic atrophy—formerly called tabetic optic atrophy—begins in the margin and advances toward the center of the optic nerves (Figs. 1 and 2).

TABLE 3

Thirteen Patients With Taboparalysis

	No. of cases
Complete optic atrophy	4
Unilateral optic atrophy	0
4 plus marginal degeneration of the optic nerves.	0
3 plus marginal degeneration of the optic nerves.	0
2 plus marginal degeneration of the optic nerves.	1
1 plus marginal degeneration of the optic nerves.	4
No marginal degeneration of the optic nerves..	4
Total	13

The degree of marginal degeneration of the optic nerves was reported from 1 plus to 4 plus. The result of the anatomic study is given in Tables 1 to 3. In Table 1 the frequency of the involvement of the optic nerves in all 40 patients, including general paralysis and taboparalysis, is shown. In Tables 2 and 3 the result is considered separately in patients with general paralysis and in patients with taboparalysis.

Discussion

In the anatomic material of 40 patients, including both general paralysis and taboparalysis, there was complete optic atrophy in 8 instances or in 20%. This figure must, of course, be disregarded as being representative of the frequency of optic atrophy in general paralysis because special efforts were made to secure permission for necropsies in these instances. The occurrence of total optic atrophy can accurately be determined by clinical examination alone and has already been mentioned to have been present in 2%.

The remaining 32 patients, however, were not selected and represented routine con-

FIG. 1.—Intermediate stage of syphilitic primary optic atrophy in a patient with dementia paralytica. There is a wide rim of demyelination in the margin of the optic chiasm.

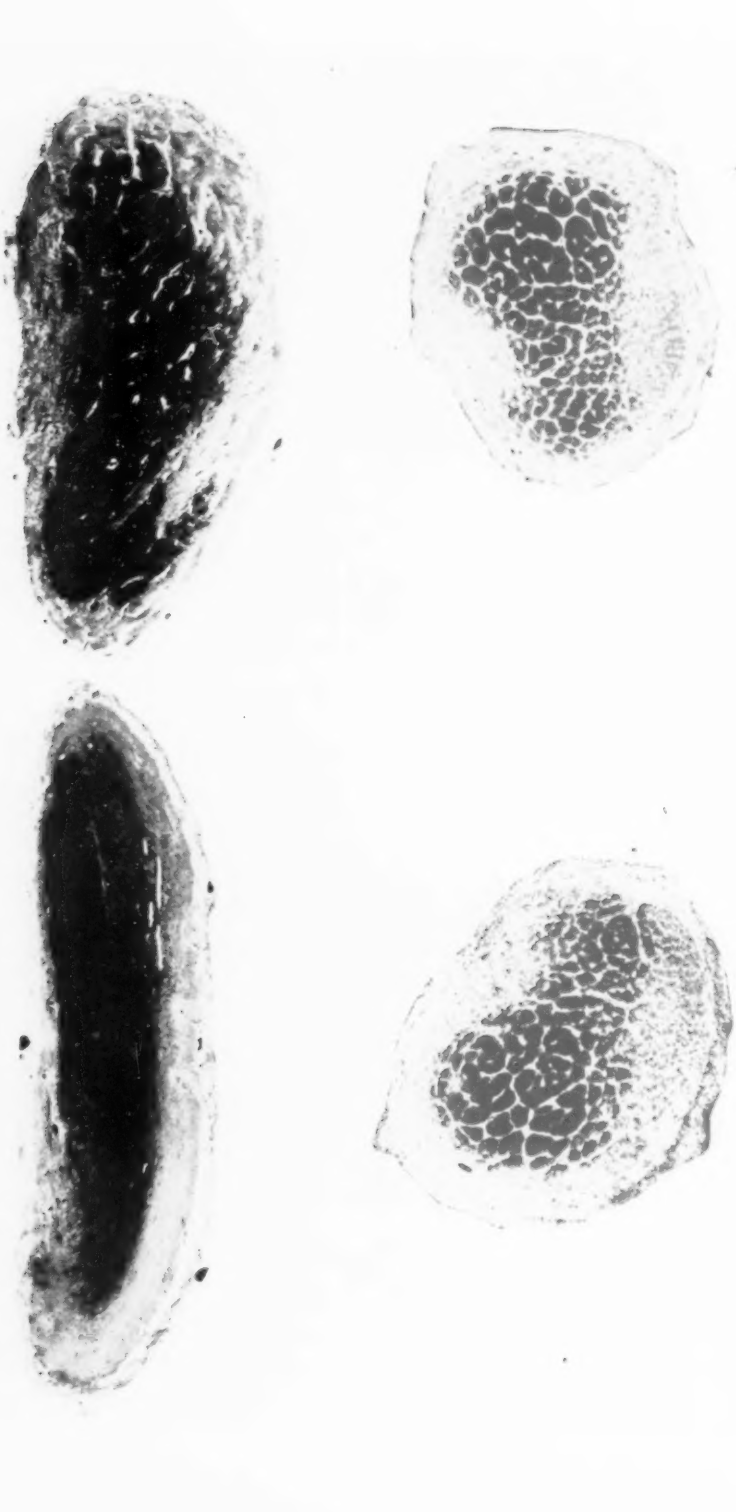

FIG. 2.—Intermediate stage of syphilitic primary optic atrophy (same case). The photograph shows the degree of marginal degeneration in both the intracranial portion (upper pictures) and in the orbital portion (lower pictures) of the optic nerves. At this stage of optic atrophy the patient is not aware of failing vision. Perimetric examination will detect the visual constriction.

secutive autopsies. From the latter group a fairly accurate idea could be obtained as to the rate with which the optic nerves in general paralysis are involved. There was marginal degeneration in 62.5%, ranging from small areas of demyelination in the periphery, reported in the tables as 1 plus, to large areas of degeneration penetrating deeply toward the center of the optic nerves, designated as 4 plus. Figs. 1 and 2 represent 4 plus marginal demyelination.

Stargardt(16) examined the optic pathways of 21 general paralytics (17 g.p. and 4 taboparalysis). There were 5 complete optic atrophies, leaving 16 cases for comparison with the 32 patients of this study. In Stargardt's group of 16 cases 56.2% revealed various degrees of marginal degeneration. This figure comes very close to my finding of 62.5%. My study was done 30 years later, showing that the prevalence of optic atrophy in general paralysis has undergone no significant change in either direction.

Is Optic Atrophy More Frequent in Taboparalysis than in General Paralysis?

On the basis of the anatomic investigation no evidence was found which would support the idea that optic atrophy occurs only in taboparalysis. Among the optic pathways of 27 general paralytics there were 4 cases with complete optic atrophy. There were also 4 cases of complete optic atrophy in the 13 patients with taboparalysis. In other words, there were twice as many instances of complete optic atrophy in taboparalysis as in general paralysis. On the other hand, in the cases revealing incipient and intermediate stages of optic atrophy, and particularly in those showing no marginal degeneration, there was no appreciable statistical difference in the two groups. The question then arises: How can in my material the greater frequency of complete optic atrophy in taboparalysis be explained? From a histologic point of view, the pathogenesis of optic atrophy is the same whether it occurs in general paralysis or in taboparalysis.

Some observers(17) are inclined to attribute the greater frequency of optic atrophy in taboparalysis and in tabes dorsalis to an unknown factor, possibly dietary deficiency. Clinically, there is no evidence whatsoever of nutritional deficiency in patients with syphilitic primary optic atrophy. However, granted that this assumption is correct, why is this unexplained factor not at work in all cases of taboparalysis? In general paralysis as well as in taboparalysis there was approximately the same number of optic nerves which showed no degeneration of nerve fibers, although some inflammatory cells were present in the pia of all the optic nerves. If one feels compelled to introduce an additional factor, why not assume the more simple concept that in some individuals the nerve fibers exhibit less resistance to degeneration than in others. From my histologic experience, however, I doubt whether the introduction of such an unknown factor is necessary at all.

It seems that the higher frequency of optic atrophy among patients with taboparalysis is more apparent than real. In part this is due to the diagnostic discrepancies as to what should be termed taboparalysis. Some clinicians are only willing to diagnose taboparalysis, if besides the absent patellar reflexes there is an ataxic gait or other tabetic symptoms such as root pains, crises, etc. In this study all cases with absent patellar reflexes with and without tabetic gait disturbance were termed taboparalysis. If two patients of taboparalysis with complete optic atrophy, who had absent patellar reflexes only, were excluded, then there would have been no difference in my material in the frequency of optic atrophy in general paralysis and in taboparalysis.

It may be added here that until recently it was the general rule that syphilitic patients with optic atrophy as the only neurologic symptom were diagnosed as tabes dorsalis on the erroneous assumption that primary syphilitic optic atrophy constituted evidence of tabes. If the criteria for the diagnosis of tabes and taboparalysis were uniform and more accurate, much of the alleged higher frequency of optic atrophy in these two types of neurosyphilis would very likely disappear. Igersheimer's(18) anatomic study seems to bear out this statement which brought the rate of optic atrophy in general paralysis and in tabes dorsalis much closer together than was hitherto the case.

Why Does Only a Certain Number of General Paralytics Develop Syphilitic Optic Atrophy?

The question why only a certain number of patients with dementia paralytica will develop an involvement of the optic nerves has greatly been advanced by the histologic examination of this large material. Although the histopathology of general paralysis is uniform in most instances, there are definite variations as to the intensity of the pathologic process in various regions of the brain. This was well exemplified by 2 cases. In the first patient, an untreated case of taboparalysis, there was an unusually marked degree of perivascular round cell infiltration in the cortex of all portions of the brain. Strikingly, there were no round cells within the optic nerves, chiasm, and optic tracts, and only a few isolated lymphocytes and plasma cells were observed in the meninges at the base of the brain. Accordingly, there was no degeneration of optic nerve fibers. In the second patient, also an untreated case of taboparalysis, there was only the slightest inflammatory process in the cortex and in the meninges of the upper aspect of the brain. The most pronounced round cell infiltration, found anywhere in this brain, was present within the optic chiasm and in the meninges of the adjacent structures. In this instance there was degeneration of all the nerve fibers of the optic pathways. The dependence of the degenerative process upon the chronic inflammation of syphilis could easily be seen. These and other instances have convinced me that the introduction of an unknown factor to explain the degenerative phase of syphilitic optic atrophy is not necessary. Léri(19), on the basis of a similar large anatomic material, has correctly pointed out that, although meningeal inflammation is constant in general paralysis, a localization in the sheaths and within the optic nerves and chiasm is not always present. This, in my opinion, is the reason why some cases of general paralysis or taboparalysis develop optic atrophy and others retain normal visual pathways.

Summary

Complete syphilitic optic atrophy in dementia paralytica, including taboparalysis, has been observed in 2%. Incipient and intermediate stages of optic atrophy are much more frequent and have been found in 62.5%, on histologic examination, of the optic pathways of 32 general paralytic patients.

There is no convincing evidence that syphilitic primary optic atrophy occurs with so much greater frequency in taboparalysis than in general paralysis, as has been reported in the literature.

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THE CLINICAL SIGNIFICANCE OF THE RORSCHACH TEST[1]

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I. Introduction

The Rorschach test appears to be one of the most significant adjuvant diagnostic devices ever introduced into the field of clinical psychiatry. It seems regrettable that its use in the fullest sense should be largely restricted to the allied science of clinical psychology. There are several outstanding reasons for this situation. Unfortunately, very few psychiatrists have written on this subject, most of the work having been done by psychologists. This has resulted in the development of a bewildering array of symbols which makes reading on this subject well-nigh impossible for the uninitiated. Another disadvantage lies in the fact that the psychiatric point of view is often lacking in writings on the Rorschach test.

We are unable, after a considerable search of the literature, to locate any work correlating Rorschach findings with a good many of the pathological reaction types. The purpose of this paper, then, is twofold: first, to assist the psychiatrist in understanding the response symbols and, second, to assay a correlation of Rorschach responses with pathological reaction types. We hope, in the latter instance, to enhance the utility of the test as an adjuvant clinical diagnostic procedure and in the former to facilitate and increase the reading of the literature on the subject.

It is our opinion that no clinical diagnosis should be based entirely on Rorschach findings. The test should be considered as auxiliary to the regular psychiatric study, just as laboratory procedures are related to physical diagnosis. In the tables (*vide infra*) to follow, it will be noted that responses are very similar in several different categories, depending upon the severity and duration of these conditions. Manifestly, then, the response pattern must be interpreted in the light of the psychiatric study. Clinical psychologists invariably require a psychiatric study before attempting interpretation. From the preceding remarks the reason is apparent.

The Rorschach tests upon which this research is predicted were done by the authors over a period of 8 years on both naval and army personnel and also civilians. They include about 3,000 cases. Our observations and conclusions are based exclusively upon responses obtained in these tests.

II. Symbols

The symbols employed in scoring Rorschach tests were developed somewhat arbitrarily. Subsequently they have become rather stereotyped. In general, the symbols are the simplest and most obvious abbreviations of the terms referred to. In the text the terms will be employed in preference to the symbols, but the latter will appear after the terms, in parentheses, with the view of familiarizing the reader with them. A glossary of response terms with their symbols and brief explanations follows:

W	Whole responses. A whole response is one which is produced as the result of seeing the blot in its entirety, rather than in any of its parts.
W-	Cut off whole. From a part of the blot, the test subject arrives at an impression of the whole. (Sometimes called W —).
D	Large detail. These are responses to obvious large parts of the whole blot.
Dr	Small or rare detail. These are responses to small details which are not such obvious parts of the whole.
de	Edge detail. Responses dealing with details around the periphery of the blot.
di	Inside detail. Responses dealing with the details inside the blot.
C	Color. Responses referring to color exclusive of form. Color shock is manifested by: 1. Avoidance of colored portions of the blot. 2. Delayed time in producing responses. 3. Disorganized responses, characterized by simply naming the colors seen.
Cn	Color naming. Simply naming the colors seen without reference to form, and when not a part of color shock.
CF	Color-form. Combined color and form response in which color predominates.
FC	Form-color. The antithesis of CF.

[1] From the Alabama State Hospitals, Tuscaloosa, Alabama.

F — Poor form. Form response in which originality is diminished. These may be bizarre. Perseveration of same form responses on subsequent cards produces F minus.
F + Good form response.
S Space response. This deals with the white spaces around and in the blot. This is interpreted as an effort to deny reality. In alcoholics it appears as a manifestation of hostility to the world.
WS Whole and space responses in combined form.
M Movement responses.
P Popular responses.
O Original responses.
A Animal responses.
Ad Animal detail. In these responses a portion of an animal may be interpreted as a whole animal. Portions of an animal or animals may be described as such when the whole animal is not seen.
H Human responses. In these responses, human forms are seen.
Hd Human detail. This is a corollary of Ad, in human responses.
K Architectural responses. Buildings, landscapes and clouds are seen. This is seen most often in card VII.
Ch Chiarascuro responses. These are visual responses which remind the subject of texture.

Some authors utilize many other terms and symbols, which we have purposely omitted from our glossary in an effort to avoid confusion. Large numbers of symbols not only serve little purpose but actually detract from the utility of the scoring system.

III. Interpretation

In evaluating reaction types, much time and effort can be saved by referring to the chart.

(A) THE NORMAL INDIVIDUAL

Whole responses (W) comprise 25 to 50% of the total number of responses. A large number of W indicates drive toward achievement. Large detail (D) should equal 60 to 70% of the total number of responses. The number of responses in the normal individual will vary. Not more than one movement (M) response should be given. Color shock is never present, and perseveration is absent. Time will vary from 5 to 40 seconds. A delay of longer than this in an otherwise normal individual should be viewed with suspicion. Color naming (Cn) is never present. Popular responses (P) are frequent, with some originals (O). Form responses should equal 50% of the total and very good form (F+) responses should approximate 85%. Animal (A) and human (H) responses should not be repeated. The usual relation of A to H is 2 to 1. The sum of rare detail (Dr), inside detail (di), edge detail (de), and space (S) responses should not exceed 10% of the whole number of responses.

(B) ORGANIC REACTION TYPE

The organic's Rorschach will of course vary, depending upon the degree and locality of the pathology, and upon the personality reaction type. In general though, structural wholes (W) will be poor because of inability to grasp a situation in its entirety. Likewise, and for the same reason, the number of responses is diminished. Lacking imagination, both movement (M) and color (C) responses are diminished. Once the organic perceives some form (F) he will repeat it in many of the following cards. In other words, perseveration is marked. Lack of confidence and blocking produce delayed time element: generally, more than one minute for the initial response. This same lack of imagination and defective recent memory produce a low popular response (P) and poor form (F) interpretation. Reiterating a former observation, if the organic sees human, animal, object, or any other indicator in the card he will repeat the observation in the remaining ones. Cerebral damage will likewise sometimes produce a bizarre effect in that things not present will be seen. We have also noted that dark shock is sometimes present especially in card IV. In summarizing, we find many factors present in other psychoses, but the entire picture of an organic Rorschach can hardly be confused because of poverty of imagination which produces the radical changes as seen on the chart.

(C) SCHIZOPHRENIA

In considering schizophrenia, we have the most difficult interpretation of all reaction types. It is obvious that a paranoid response will differ from a catatonic or hebephrenic record. The authors believe,

however, that the clinical Rorschach interpretation of schizophrenia, regardless of the type, produces a series of responses which are fairly typical. Consulting the chart, we find that there are contaminated and structurally poor whole responses. The catatonic will give these responses, as will the hebephrenic. Regarding the number of responses, we find that there are many in the paranoid but few if the record is flat (catatonic type). In our experience, movement (M) is low or absent except in the paranoid type. Color responses are the most confusing of all. The paranoid will produce many pure color (C) and color form (CF) but few or no form color (FC) responses. The catatonic will see little or nothing and the hebephrenic's interpretation is valueless because of marked dissociation of affect. The catatonic, pursuing one line of thought, will demonstrate marked perseveration in some indicator, *e.g.*, animal detail (Ad).

In regard to the time element, it is of course very rapid in the paranoid, except when affective blocking is prominent, but will be greatly increased in the catatonic. We have observed also that the catatonic, if perseveration is not marked, may refuse cards. Often the schizophrenic, especially the hebephrenic, will simply name colors without any attempt to describe them (Cn). Popular responses (P) are diminished and form is poor (F−). Catatonics, particularly, will show stereotypy responses. We have found another peculiar factor in the routine of responses, in contrast to some psychologists, in that there is emphasis on rare detail (Dr) followed by whole responses (W). In other words, the schizophrenic will first select rare details (Dr) and then attempt to interpret the whole (W). Color shock, as evidenced by avoidance of color and delayed reaction time to color, is present in nondeteriorated schizophrenics.

(D) MENTAL DEFECTIVES

Like the schizophrenic, the mental defectives produce poor whole responses (W) as well as vague and cut-off wholes (W). The difference lies in the fact that the whole response (W) in the mental defective is often confabulatory. The entire record is flat because of lack of imagination and the fact that his social contacts are markedly restricted. Consequently, the number of responses is low, especially those of movement (M). On the other hand, the mental defective is generally intrigued with color and there will be many pure color (C) and color form (CF) responses. Perseveration is, of course, increased and initial time response is delayed due to lack of imagination. Sometimes too, a color (C) will be named without attempting interpretation of the form (F). F+, or superform, is low as the mental defective does not attempt to evaluate the cards except in a stereotyped manner. Details are generally omitted.

(E) PSYCHONEUROSES

Before considering the Rorschach of a psychoneurotic, we should first attempt to understand his background and viewpoint in this application. Tension resulting from conflict prohibits his making adjustment to his environment. As noted by most psychiatrists, he will overemphasize one adjustment factor to the complete exclusion of all others. He is therefore incapable of obtaining the balance of factors which we call normalcy.

The neurotic presents an aberrant record. Lacking drive he will show whole (W) responses of less than 30% but his details, large detail (D), rare detail (Dr), edge detail (de), and inside detail (di), will be 70% or better. Anxiety neurotics will give few responses, but in obsessive compulsives it is the experience of one of us (C. E. J.) that the number is greatly increased. He recorded 442 responses in one such case. In considering this type, the increased number of responses is obviously due to the perfectionistic trend. The opposite holds true for conversion hysteria and neurasthenia. Movement is increased in obsessive-compulsive neurosis while decreased in conversion hysteria and anxiety neurosis. Color reaction is generally neutral except in the obsessive-compulsive where there is an uncontrollable response to color (C) with many pure color (C) and color form (CF) responses. Perseveration is often seen in the anxiety neurotic due to restriction of imagination. The time element shows some variation. It is slow in psychoneuroses, with

the single exception of the obsessive-compulsive type. In the latter it may be slow if perfectionistic trends are marked.

Color naming (Cn) is sometimes present in the psychoneurotic and is quite common in the obsessive-compulsive type. On the other hand, popular responses (P) are diminished in the obsessive-compulsive type but increased in the other psychoneuroses. These diametrically opposed phenomena constitute an important differentiating point in distinguishing the obsessive-compulsive from the other psychoneuroses. The psychoneurotic, in general, will also produce several form color (FC) responses in which form is predominant over color. Sometimes, too, form color (FC) responses are present in which the subject will interpret the blot as an architectural landscape (K). One example is seen in card VII where the interpretation of clouds is often given. In the obsessive-compulsive group, animal and human detail will exceed animal and human whole responses due to preference for small detail and to the characteristic perfectionism. For the same reason, we find that rare detail (Dr), inside detail (di), edge detail (de), and space (S) interpretations exceed the normal of 10%. Anxiety neurotics occasionally completely refuse a card. Marked restriction of imagination is usually seen here. This is evidenced by a reduced number of responses for the whole test. A peculiarity of the obsessive-compulsive type is the rigidity of interpretation. This individual will usually follow a definite routine in most of his responses. These, in brief, constitute the principal characteristics of the psychoneuroses. Their differentiating points are easily ascertained by reference to the chart.

(F) ALCOHOLISM

The alcoholic reveals personality changes which are particularly evident in the last 3 cards. On these he will produce whole responses (W), indicating aggressiveness. This may not necessarily change the ratio of the number of responses on the last 3 cards to the total number of responses.

$$\text{Cards}\ \frac{\text{VIII, IX, X}}{\text{Total responses}} = \text{R}.$$

Rather, it indicates an extreme effort to interpret the highly colored cards as a whole. This differs from all other psychoses and is simply a defense mechanism which reveals itself in hostility to the world in general. The total number of responses varies. Movement (M) may not be present in the Rorschach pattern of the chronic alcoholic but will be seen in the short interval after delirium when the subject is able to cooperate. We have never been able to determine the absence or presence of perseveration as characteristic. While it is true that there is often a preponderance of animal or human indicator, it does not occur frequently enough to be called perseveration. Severe alcoholic psychoses may show a sufficient number of animal and human responses to represent compulsion. Color response (C) and color naming (Cn) are either absent or very rare. The time response also varies with the severity of the symptoms and the personality reaction type. The number of popular responses (P) will vary. Form is generally poor (F−) because of preponderance of animal (A) and human (H) forms seen. When whole detail (WD) and rare detail (Dr) are poorly seen, it represents confabulation as seen in Korsakoff's syndrome. This confabulation represents the alcoholic's effort to force things to comply with his viewpoint, regardless of the consequences.

(G) MANIC-DEPRESSIVE PSYCHOSES

1. *Manic Phase.*

The literature on manic-depressive psychoses in this connection is remarkable for its paucity. We are recording our observations and results and earnestly hope that other investigators will add to the Rorschach interpretations. We have, therefore, been very conservative and have revealed only the most positive features of the test.

Whole responses in the manic are increased while the number of responses is diminished. This will therefore give a high percentage of whole responses (W), sometimes equal to or very near the total number. Movement responses (M) are more than the normal of one. We have not arrived at any definite conclusions regarding color (C). Initial time of response is generally of short duration. Color naming (Cn) is absent.

SUMMARY OF

	Whole responses W and D	Number of responses	M Movement	C Color	Perseveration
Normal individual.	W is 25% to 50%. Large number of W indicates drive for achievement. 60% to 70% large detail D.	Varies	Not more than one.	No color shock.	Absent.
Organic.	W —.	Total number of responses less than 15.	Not more than one M.	Few C.	Marked.
Schizophrenia.	Contaminated W responses. Structurally poor wholes.	If record is flat, catatonic number of responses few.	M responses are few except in paranoid.	Paranoid type may have C and CF but few FC.	Marked in catatonics.
Mental defective.	Poor whole responces. Vague structural wholes. W is confabulatory.	Varies, but generally diminished.	Movement is low.	Many CF and C responses.	Increased.
Psychoneuroses.	(1) Less than 25% W. (2) More than 75% P.	Few responses in anxiety and conversion hysteria. Greatly increased in obsessive compulsive.	Increased in obsessive compulsive. Decreased in conversion hysteria, neurasthenia, and anxiety.	Oftimes there is an uncontrollable response to color, especially in obsessive compulsive.	Often seen in anxiety.
Alcoholics.	W responses on last 3 cards indicates aggressiveness.	Varies.	Varies.	Absent.	Depending on severity of compulsion.
Manic-depressive, manic phase.	W increased.	Diminished.	More than one.	.....	.....
Manic-depressive, depressive phase.	W —. Wholes of poor form.	Diminished.	Absent.	Diminished or absent.	Frequent.
Involutional psychosis, melancholia.	Diminished.	Diminished.	Absent.	Diminished or absent.	Frequent.
Involutional psychosis, paranoid type.	Poor.	Compares well with normal.	More than one.	Disturbed by color. CF at expense of FC.	Infrequent.

RESPONSES

Initial time	Cn Color naming	P Popular responses	F minus and F plus	A Animal H Human	Miscellaneous.
Normal 5" to 40".	Absent.	Frequent with some originals.	F minus 50%. F plus 85%.	No repetition. Usual relation of A to H is two to one.	Sum of Dr, Do, di, de and S should not exceed 10%. A balance between original and popular responses.
More than one minute.	No more than one.	Below 25%.	Poor F — (F minus). Low F plus.	Repetition of the indicator.	Lack of confidence. Dark shock. See things not there.
Delayed in catatonics. Rapid in paranoid.	Present.	Much decreased.	Poor form F —.	Stereotypy of responses, particularly in catatonics.	Routine of responses is Dr — W — D, with emphasis on Dr and W. Confusion of sequence. Color shock is present in non-deteriorated individuals.
Delayed.	Increased.	Decreased.	Low F plus.	Stereotyped responses.	Omission of parts of card.
Generally slow.	Sometimes present, especially in obsessive compulsive.	Increased except in obsessive compulsive.	Several FC or FK.	Ad and Hd exceed A and H.	More than 10% Dr, di, de, S. Rigidity of interpretation, especially obsessive. Sometimes there is complete refusal, especially in anxiety.
Varies, depending on severity.	Rare.	Varies, depending on personality reaction type.	Many F —, persistent interpretation.	Increase of H or A over normal.	Over-glorification of female; vases, urns, etc. Where WD and Dr are poorly seen it indicates confabulation, as in Korsikoff's syndrome.
Generally of short duration.	Absent.	Fair.	Fairly good.	Increased.	Repressed sexual responses with attention to lower details. D increased. Dr decreased.
Prolonged.	Usually absent.	Poor.	Poor.	Absent.	General reduction of W, D, and C responses.
Prolonged.	Usually diminished.	Diminished.	Poor, varying with amount of depression.	Diminished.	General poverty of imagination. Flat record. Bizarre responses.
Slightly diminished.	Absent.	Fair.	Good.	H increased over A.	Paranoid productivity diminished by anxiety constriction.

The number of popular responses (P) is generally fair. There is an increase in animal (A) and human (H) responses. Large detail (D) is increased and rare detail (dr) decreased. Repressed sexual responses are noted by avoidance of any pelvic or phallic interpretation or attention to the lower details of the cards. Form (F) is usually fairly good.

2. *Depressed Phase*

In the depressed phase of manic-depressive psychosis, whole responses (W) may be increased but they are of poor form (F−). The number of responses is diminished and movement (M) is absent. Color (C) and color naming (Cn) are usually absent, indicating depression and disinterest in environment. The number of popular reponses is poor. Animal (A) and human (H) interpretation are absent. Form is poor (F−) because of inability to interpret anything with imagination. If the reader will refer to the chart, he will observe the points of similarity in the manic-depressive psychoses and also the differentiating points between the manic and depressive phases.

(H) INVOLUTIONAL PSYCHOSES

1. *Melancholia*

Involutional psychosis is essentially a descriptive term. It may manifest itself in two phases, either a state of melancholia or of paranoia. In the chart it will be found that the Rorschach responses of manic-depressive psychoses, depressed type and involutional psychoses, melancholia type are practically identical. Likewise, the experienced psychiatrist may label the same case involutional melancholia and manic-depressive psychosis, depressive type. In this instance, the background, age of onset, history of climacteric, and many other factors must of necessity take precedence over the Rorschach. Since this is not research on psychiatry, but on the interpretation of the Rorschach record, the discussion will be confined to an investigation of the results. One possible differentiating point is the whole responses (W) which are diminished in involutional melancholia but may be normal, but of poor structural wholes (W−) in the depressed manic. The number of responses, movement (M), and color (C), are all diminished. Perseveration is frequently observed and initial time responses prolonged. Popular responses (P) are diminished and form is poor (F−) depending upon the depth of depression. Animal (A) and human (H) responses are diminished. In general there is poverty of imagination with a flat record with occasional bizarre responses.

2. *Paranoid Type*

The paranoid involutional comprises an entirely different picture. Whole responses are poor (W−) but the total number is not necessarily decreased. Likewise the total number of responses compares well with normal. Time responses are diminished. There is more than the normal number of movement responses (M). The paranoid involutional is disturbed by color (C) but actual color shock is not present. He will show color form (CF) responses at the expense of form color (FC). Color naming (Cn) is absent. The number of popular responses (P) is fair. Form response in general is good, but not excellent. Human (H) responses are increased over animal (A) interpretations, a reversal of the normal record. Paranoid productivity with involutional psychosis is diminished by anxiety constriction. It will be seen in the chart that the paranoid type of schizophrenia cannot possibly be confused with the paranoid type of involutional psychosis.

IV. CONCLUSION

It will be noticed that the authors have not laid overemphasis on sexual responses. These are manifested by particular attention to the lower details, interpreted as portions of the female pelvis or to phallic symbols or to their complete avoidance (sexual repression). This is because they occur generally in behavior problems and correctly belong in the realm of clinical psychology. Repressed sexual responses are occasionally encountered in the manic type of manic-

depressive psychosis (particularly when the patient is recovering and is unsure of himself) and more rarely in psychoneuroses. Likewise, we have not attempted to interpret the degree of mental defect because this can be ascertained only by a psychometric test and not by the Rorschach. We do believe from our findings that the Rorschach test can be of tremendous value in psychiatry but, as previously stated, should be nothing more than a useful aid to the psychiatrist in his diagnostic investigations.

THE ATTITUDE OF THE STATES TOWARD COLLECTING FEES[1]

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The advance in medical knowledge has led to more complicated ideas of therapy which in turn have led to increase in the cost of medical care. The need for better medical care has been felt throughout the medical world, but especially in the hospitals concerned with the care of the mentally sick. These hospitals arose as asylums in the beginning of the 19th century, at first supported by the county, and then by the state. They became known as state hospitals because of their support from state funds, and for years they have presented the outstanding examples of the effects of state medical control. Now with the growing demand for more and more therapy as well as better results from therapy, the funds appropriated by the state have been found constantly inadequate, so there has been a trend away from full state support toward state support supplemented by private fees.

In order to gain insight into the attitude of the different states of the union regarding the business of charging fees to state hospital patients, questionnaires were sent to the mental hygiene authority in each state and the District of Columbia. Replies came from all. It was estimated what percentage of the funds spent on state hospitals was represented by fees and what percentage of patients paid fees of one kind or another. It was found that different states charged different fees and had different methods of collecting them. Some were not allowed by law to collect (see Table 1); some were allowed but made no extensive effort to do so; in some the fees were handled by the counties; and some made a definite effort to finance their state hospital program through direct charges. In order to estimate the financial standing of the state hospital system of each state so that those charging fees could be compared to those not charging, the cost of care per patient per diem was taken from the 1945 census as given by the Bureau of the Census in Washington, D. C., in Bulletin Series M.P. No. 13 issued April 3, 1947. Thus we were forced to compare per diem costs of 1945 with collections for 1946, producing a definite discrepancy.

TABLE 1

THE FIVE STATES IN WHICH COLLECTION OF FEES IS NOT PERMITTED

	Percentage of total funds from fees	Percentage of patients paying fees	Cost of care per diem per patient	Charge per week
Illinois	0	0	$1.31	$0.00
Georgia	2.3	..	0.65	5.00
Oklahoma	1.2	2	0.60	6.25
Tennessee	10.0	5	0.62	8.25
Virginia	6.0	2	0.76	5.25

The collection of fees in Virginia is confined to voluntary patients or to patients going to a state-supported private sanitarium where fees are charged. The attitude of Illinois is that there should be no collection of fees, but it is better to have all the financing of the state system come directly through the state. That state has a high per diem cost of $1.31 per patient. Wisconsin, a state which has been collecting fees, writes as follows: "Two years ago the legislature passed a bill relieving the patients and their families from paying for the care of tuberculous patients, and there are those that believed the same conditions should prevail for the mentally ill, the theory being that patients would be encouraged to receive treatment and to remain in the hospital until it was safe for them to leave. Where there is financial pressure on the relatives, the tendency is to remove the patient too early or to refuse to

[1] Read at the 103d annual meeting of The American Psychiatric Association, New York, N. Y., May 19-23, 1947.

send the patients to the institution when they really need help."

Table 2 shows those states where the fees are handled through the counties and no definite estimate can be made of their value in state financing.

Table 3 shows the financing of 18 states that are allowed by law to collect fees, but no definite effort is made, so that their returns are small.

The practice in Rhode Island would seem to be typical of this group. After admission

TABLE 2

STATES IN WHICH FEES ARE HANDLED THROUGH COUNTIES

	Percentage of total funds from fees	Percentage of patients paying fees	Cost of care per diem per patient	Charge per week
Iowa	..	..	$0.73	$0.00
Maryland	..	..	0.73	7.50
North Dakota	..	..	0.86	7.00
South Dakota	..	..	0.78	6.25
West Virginia	..	..	0.74	14.00

TABLE 3

STATES THAT ARE ALLOWED TO COLLECT FEES BUT MAKE NO DEFINITE EFFORT

	Percentage of total funds from fees	Percentage of patients paying fees	Cost of care per diem per patient	Charge per week
Alabama	Negligible	10	$0.70	$7.50
Arkansas	3.0	4	0.66	4.00
Florida	..	4	0.92	8.25
Idaho	2.97	5	0.94	8.75
Indiana	..	10	0.70	5.00
Kentucky	1.00	3	0.54	7.50
Louisiana	..	2	0.74	7.00
Maine	..	10	1.00	10.00
Michigan	..	6.7	1.43	10.57
Mississippi	2.0	2.5	0.63	4.25–12.50
Missouri	5.0	5.0	0.84	10.00–12.50
Montana	7.0	16.0	1.00	7.00
New Mexico	2.0	13.0	1.00	7.00
Ohio	..	15.0–20.0	0.84	5.50
Rhode Island	..	15.0	0.69	7.00
Texas	10.0	10.0	0.60	5.00
Vermont	..	16.0	1.03	9.00
Wyoming	..	7.1	0.81	6.25

North Dakota and West Virginia have a right by law to enforce collection of fees, but the Mental Hygiene authority was unable to state percentages obtained by charges by the counties. The case of South Dakota is typical of this group. The state legislature provides money for the operation and running of the hospital. The money is taken from the general state funds. Counties are charged at the rate of $25 per month for each admission from that county. A voluntary patient pays $25 a month which the county collects from him or his estate if he has one; if not, the county pays the bill. The money collected goes to the general funds of the state. It is not definitely allocated to the hospital system.

of the patient to the hospital, an attempt is made to obtain the maximum rate of board of $10.50 per week. If the maximum rate cannot be obtained, a staff investigator of the State Department of Public Welfare determines the ability of the patient to pay or the liability of the estate if any, and a charge is made according to the ability to pay. The collections go into the general state funds and are separate and apart from the state hospital funds.

Table 4 lists those 21 states in which a definite effort is being made to collect fees so that the returns represent a sizeable portion of the hospital income.

It is interesting to note that 20 states out

of the 49 [2] have a per diem per patient cost of $1.00 or over, and of the 20, 14 are found in Table 4 while two others on the list have 97 cents and 99 cents as their daily expenditure.

The special conditions found in Delaware, North Carolina, and South Carolina add interest to this group. Dr. Tarumianz, Superintendent of the Delaware State Hospital, writes as follows: "The laws of the state of Delaware permit Delaware State Hospital to charge immediate relatives of any patient admitted to the hospital the amount necessary for the care of such patients. They do not specify that the hospital should not charge more than the average cost of other patients. We have established a procedure which has been accepted very graciously by the people of the state of Delaware. 'Everyone who can pay anything at all for the care of a patient should do so.' Therefore we have charged as little as 50¢ a day and as much as $9.00 per day. We have a contract with the Blue Cross Group Hospital Insurance Company and other insurance agencies. They pay us for the care of the patients who carry such insurance policies, the same amount as they pay in any accredited hospital. Through this agreement we receive $13.00 for the first day and $5.50 per day for the consecutive 29 days. For the second period of 30 days they pay us $2.50 a day. Under the contracts for veterans admitted to this hospital, the Veterans Administration pays $5.00 a day per capita. The average monthly income from paying patients is $15,000 to $16,000. This is about 26% or 27% of the total expense of the maintenance of the patients. This amount is paid by about 17% of the population of the hospital. We are expecting an increase in our income to $22,000 per month. This additional income affords us to have better facilities for all. According to the laws the money obtained from the patients is deposited in a special account fund and used for no other purpose except maintenance and fees including medical service. May I also state that this method has improved our relationships with the people. The hospital was not considered a place for the indigent people. People of means, upper and middle class, are gladly sending their sick ones to our hospital for care and treatment. It has also helped to eradicate the stigma usually connected with mental hospitals."

TABLE 4

STATES THAT MAKE A DEFINITE EFFORT TO COLLECT FEES

	Percentage of total funds from fees	Percentage of patients paying fees	Cost of care per diem per patient	Charge per week
Arizona	6.0	14	$1.29	$5.16
California	..	25	1.03	2.50–10.00
Colorado	8.1	..	1.25	..
Connecticut	..	45	1.16	5.00
Delaware	27.0	17	.97	3.50–56.00
Washington, D. C.	13.9	15.5	1.95	24.85
Kansas	18.0–20.0	35	.70	5.00
Massachusetts	8.5	10	1.21	1.00–10.00
Minnesota	14.3	..	.78	7.00
Nebraska	14.63	16.81	1.00	8.15
Nevada	7.0	..	1.00	5.00
New Hampshire	..	21	1.30	8.75
New Jersey	6.5	40	1.23	14.00
New York	12.0	23	1.40	1.25–20.00
North Carolina	4.7	5.7	.81	7.50–10.00
Oregon	10	20	.68	6.25
Pennsylvania	15	30–35	1.00	7.21–11.97
South Carolina	..	..	.69	7.00
Utah	20	..	1.05	7.50–12.50
Washington	..	25	.99	8.33
Wisconsin	..	25	1.54	5.00–14.80

North Carolina requires that the clerk of

[2] Includes District of Columbia.

court will act as a judge to make commitments in the county, furnish information in regard to the property of the person being committed, and of assets of other persons liable for the patient's care. A questionnaire regarding these matters is printed on the back of the commitment papers and must be filled out along with them. Persons considered liable for the care of the patient include the husband or wife of the patient, and the parents of any minor children. Charges are limited somewhat, because the constitution exempts the home and an amount of $1500 from debt. However, such exemption does not continue after death, and collections are often made from a patient's estate whether or not he remained in the hospital until the time of his death. Provision is also made that, if a person should later acquire money, this would be liable to charges. The clerk of court ordinarily informs the hospital of the death of one of the patients, and the collections are made either by the money being sent to the business office of the hospital concerned or, if a claim has to be filed, it is done in the attorney general's office by someone who receives special compensation for this and who also investigates the finances of the family. At the present time white patients are charged $40.00 a month, colored patients $30.00 a month, and any or all of this that they do not pay is held as an indebtedness against them or their estate. In the state of North Carolina alcoholics are not admitted to the hospital unless their family or the county from which they come will pay for them. The charge for a 2-month period for alcoholics is $120 to be paid by the patient or his family, and $80 to be paid by the county. In the state hospital at Raleigh for the year 1945-1946, fees were paid by 11.5% of the patients and covered 7.3% of the cost

Dr. Coyt Hamm, Superintendent of South Carolina State Hospitals, writes as follows: "The South Carolina state hospital operates on a budget system. The appropriation by the General Assembly is made in a lump sum with the proviso that the hospital is permitted to spend in addition to that amount all cash revenues. This arrangement is an admirable one and gives the Board of Regents the opportunity of using available funds for the best interests of the mentally ill for whom we are caring. The statute provides that all persons financially able to do so pay at the regular rate of maintenance which rate is set by the Board of Regents determined by the economic conditions of the state at the time. The rate of pay closely approximates the daily per capita cost of the operation of the hospital, and at the present time we are collecting $1.00 a day."

From Table 4 it can be seen that the percentage of total funds obtained from fees varies from 0-27%. The average for 26 states is 8.8%. The percentage of patients paying something in the way of fees varies from 0-45 with an average for 38 states of 14.3. Taking only the 21 states that we have listed as making a definite effort to collect fees, we find that the average percentage of funds collected is 13, and the patients paying something average 24.5%.

Methods of collecting fees vary from state to state, but can be divided into categories under the heads of (A) fees charged, (B) agency making and collecting the fees, and (C) use of fees.

(A) *Fees Charged:*
1. None.
2. Only voluntary patients.
3. A set amount per week or per month as determined by law.
4. The per diem cost.
5. Same as general hospital.

The large majority fall in the group that charges a set amount, or the per diem cost. Only the state of Delaware and a few so-called "treatment" hospitals can charge as a general hospital does.

(B) *Agencies Setting and Collecting the Fees:*
1. The judge at the time of commitment.
2. A special commissioner or department under the State Welfare, State Collector of Internal Revenue, or Health Department, who investigates the financial condition of the patient and his relatives and determines the fees, or recommends fees to judge.
3. The state hospital superintendent or business manager who talks with the patient and his family and agrees on a fee.

The best arrangement is probably that of the District of Columbia. Despite the high cost of $3.55 per day for all their patients, they managed last year to collect 13.9% of

this from patient's fees. The main feature of their setup seems to be a "special financial unit" which makes a detailed investigation of each patient's personal assets and of the legally responsible relatives, then recommends to the court what the patient or his relatives can pay. If the patient has an estate, a committee of one is usually appointed to handle this estate, the committee usually being a relative of the patient. The court makes the final decision as to what amount should be paid from the estate of relatives, and these payments are made to the collector of taxes and go into the general funds of the District. It is a policy of the special financial unit to accumulate sufficient funds first for burial of the patient and also to set aside a reasonable sum that the patient may draw upon for his personal use and benefit at the hospital. This latter amount usually averages $5.00 a month, depending on the patient's assets, age, etc. Any amount remaining after payment of administrative cost is then applied as fee. A letter from this special financial unit says: "As a whole the attitude is very good, and the courts and attorneys are very cooperative, as are most of the relatives. The public is gradually being educated to the need of everyone paying his own way in our institution as far as his assets or income will permit."

(C) *Use of Fees:*

1. In most states the fees are turned into the general fund of the state while the hospital is supported by a direct appropriation from the state treasury. How much effect the percentage of fees collected has on the appropriation received is not evident. Kansas collects 18% of hospital funds from fees and has a per capita per diem rate of 0.70.
2. The fees may be paid directly to the hospital and used in any way it sees fit. This is done in South Carolina.
3. The fees go directly to the hospital but are set up as a special fund that can be used only for the care and treatment of patients. This is done in Delaware.

We have no more accurate method of rating the quality of care and treatment given by the various states than from the per diem expenditure, personnel employed, and the like. There does not seem to be a valid correlation between the percentage of fees collected and the quality of care given, if we take the 48 states as a whole. However, closer examination shows that the states which are fairly effective in collecting fees can be divided into 2 groups with considerable differences. In the one group are those states whose per diem cost is low—say 70-90¢—but who collect say 15-25% of this from fees. On the other hand, there is that group of states where the per diem expenditure is greater—say $2.00—and which collect no larger percentage of their funds from fees or maybe even less—yet they spend more on the patient, as in Washington, D. C. In this group it would seem that the charging of fees represents a rather large and necessary part of their financial support and is a real factor in enabling them to strive for better care and treatment.

It is true, however, that so long as charges cannot exceed per diem cost these states with a high per diem cost will not be able to increase the percentage of funds collected from fees. The widespread practice of paying fees into the general funds of the state must decrease their direct benefit to the state hospital system. Delaware is the one state that has removed both the above limitations and the results seem very encouraging.

The following conclusions are justified:

(A) Most states can and do collect fees and the percentage of funds derived from this source depends on:

1. A sound law;
2. An effective collecting system;
3. The general economic condition of the state.

With a good law and organized effort a state may expect to collect 10-20% of their total funds from this source. Furthermore, they should be able to collect some fees from about 20-40% of their patients. The example of Nevada and Arizona should be given here. In these two states renewed interest in collecting fees under existing laws has resulted in considerable increase of income. Sidney J. Tillim, M. D., superintendent of the Nevada Hospital for Mental Diseases, writes as follows: "As for the paying patients, up to 1946 there was a time when the requirement was $20 a month for those

able to pay. However, in actual practice a very small fraction of those able to pay did so. The income was approximately 2% of total operating cost. More judicious enforcement of the requirement raised the income to approximately $1,000 a month during 1946, and it is gradually growing in amount. We estimate this source of income should amount to about 10% of the operating cost of the hospital. We feel it is reasonable and a fair requirement that those patients and relatives in a position to pay for services should at least pay the actual cost per diem per capita because of direct benefit derived for the nominal cost compared with similar services in private hospitals. In Nevada we do not have any private hospitals and the cost of taking patients into neighboring states is prohibitive for most cases." In Arizona collection of fees was increased by $12,000 in 1946 because a special assistant to the Business Manager was appointed for that purpose.

(B) A rather small group of states have raised the quality of care and treatment given in their hospitals to a higher level; the charging of fees has been a necessary and effective factor in this endeavor.

(C) There is a trend to go from a purely state-supported state hospital system to one more nearly approaching that of the general hospital with state support. Only two states, Illinois and Wisconsin, stand against this trend. At a time when the trend in medicine in general is toward greater state support, this movement in the opposite direction in some mental hospitals is significant. It has come as a part of a general movement for more effective treatment.

Furthermore, for those interested in improving the fee setup in their states, this survey has shown that the following factors are necessary if the maximum benefit to the treatment of the mentally ill is to be gained from the charging of fees:

1. A sound law allowing fees to be charged and collected, and in certain cases to exceed the per diem cost, especially for voluntary patients.

2. A special department or commission to investigate the financial status of each patient and his family and advise a fair fee in each case.

3. Provision that the funds collected from fees go directly to the care and treatment of the patients.

4. Continued attempts to raise standards of treatment and improve public relations so the general public may come to a better appreciation of the needs and problems involved in the treatment of mental illness.

CASE REPORTS

TRANSIENT AURICULAR FIBRILLATION COMPLICATING ELECTRIC SHOCK THERAPY[1]

WALLIS L. CRADDOCK, M. D., AND HOWARD P. GILBERT, M. D.,

Fort Logan, Colo.[2]

Reports of cardiovascular complications are very rare in electroshock therapy according to Kalinowsky and Hoch(1), who state, "In our own material we do not remember any case developing cardiac symptoms, and the absence of important electrocardiographic changes during electroconvulsive therapy make them improbable." In metrazol treatment, cardiovascular complications, especially auricular fibrillation, are occasionally encountered. Hoffman(2) reports this as having been seen once in a series of 1296 metrazol shocks. A review of the literature fails to mention a case of auricular fibrillation in electroconvulsive therapy.

REPORT OF CASE

A 52-year-old white male was admitted to the Veterans Hospital, Fort Logan, Colo., on March 21, 1947, complaining of weakness, headaches, fatigue, and a feeling of depression for about 7 months. For 3 or 4 weeks prior to admission he was completely bedridden with vague, neurasthenic complaints. He presented the picture of severe depression, yet he was found to be in good physical condition, fairly well nourished and well developed. His heart was normal in size and regular in rhythm. The blood pressure was 114/70. A thorough examination, including neurological study, failed to reveal pathological findings.

Laboratory findings, including a complete blood count, sedimentation rate, spinal fluid examination, serology, blood NPN, and urinalysis, were negative. X-ray studies of the lungs, kidneys, gastrointestinal tract and skull revealed no pathologic conditions. Cardiac clearance was given, and the cardiologist stated, "Electrocardiographs of April 1, 1947 and April 10, 1947 show no significant differences between the tracings. Low voltage QRS in lead I and slight upward deviation of ST in leads I, II, and III appear, but these records may be considered within normal limits." (Fig. 1.)

After psychiatric consultation, a diagnosis of depression of the involutional period with both autogenous and reactive features was made. Electroconvulsive therapy was recommended, and the patient was transferred to the neuropsychiatric section after his thorough work-up on the medical ward.

The first electroconvulsive treatment on April 16, 1947, using 110 volts for 0.3 sec., resulted in a petit mal reaction. After allowing for more normal breathing, the patient was given a shock using 110 volts for 0.5 sec. and the desired grand mal convulsion resulted without cardiac complications.

On April 18, 1947, a second treatment using 120 volts for 0.5 sec. resulted in a grand mal convulsion. It was noted that the irregular heart action of auricular fibrillation had appeared. An electrocardiogram was immediately taken. It showed a ventricular rate of 110 with the picture of auricular fibrillation (Fig. 2). No specific treatment was instituted, and the fibrillation spontaneously terminated in normal rhythm in about 3 hours.

During the remaining 4 treatments the patient was followed very closely for possible cardiac collapse. During these treatments he received the "double shock" method using 120 volts for 0.5 sec. It was noted on April 21, 1947, that there was absolute cessation of the pulse for about 10 seconds after the electroconvulsive treatment, but regular rhythm was resumed after this period of apparent cardiac arrest. Treatments on April 23, 25, and 29 revealed no further irregularities. It was deemed wise to terminate the electroconvulsive therapy after the 6 treatments. The patient gained weight, became bright and cheerful, and had no further complaints as of those on admission to the hospital. He was discharged on May 6, 1947, with a guarded prognosis, especially in view of a rather turbulent domestic situation. It was agreed that he should be recalled after 3 months for a check-up of his physical and mental condition.

On August 12, 1947, he was seen as an out-patient. He appeared in excellent health and stated that he had felt perfectly well since discharge from the hospital and had had no recurrences of his depression even though his domestic relations continued unimproved.

A thorough cardiac and neurological examination

[1] Veterans Administration Hospital, Fort Logan, Colo.

[2] Published with permission of the Chief Medical Director, Dept. of Medicine & Surgery, who assumes no responsibility for the opinions expressed or conclusions drawn by the authors.

From the Psychiatric Section of the Medical Service, Veterans Administration Hospital, Fort Logan, Colo.

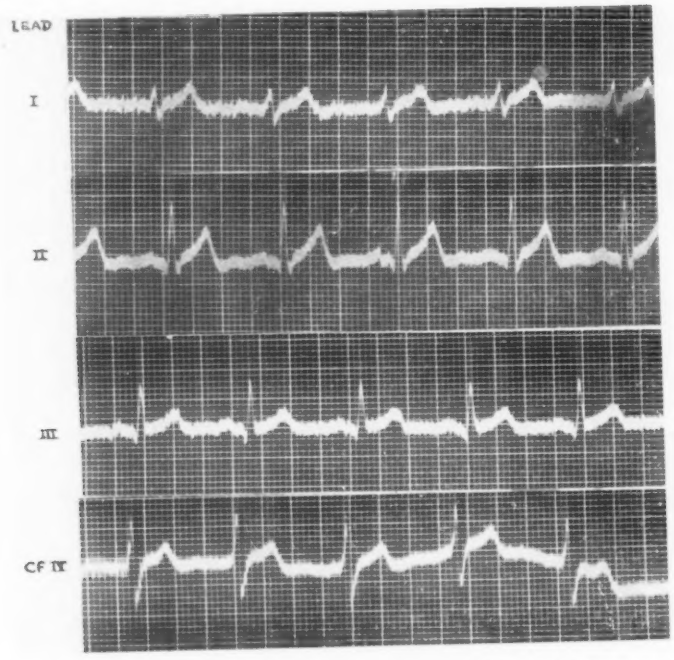

FIG. 1.—Electrocardiogram of 1 April 1947, showing findings within normal limits. ECG repeated 10 April 1947 with similar picture prior to institution of electroconvulsive therapy beginning 16 April 1947. Cardiac clearance given in view of thorough cardiac work-up.

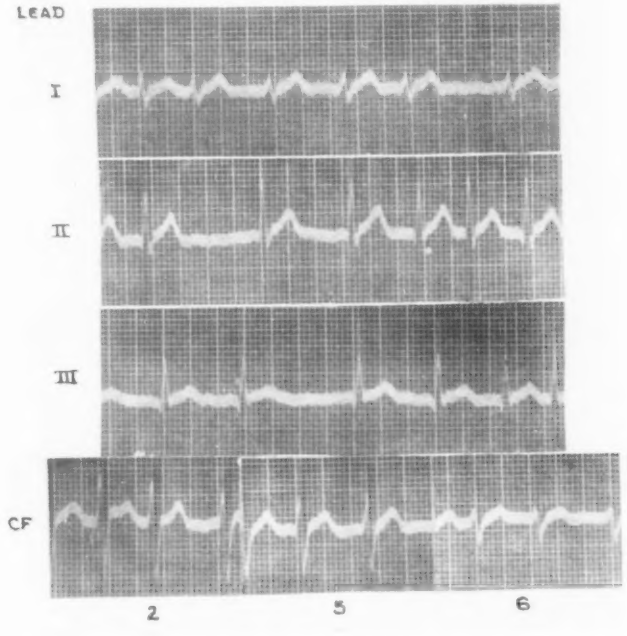

FIG. 2.—Electrocardiogram of 18 April 1947, taken shortly after patient received second ECT, showing auricular fibrillation, which terminated spontaneously about 3 hours later.

FIG. 3.—Electrocardiogram of 12 August 1947, approximately four months after the EEG of 18 April 1947 which showed the auricular fibrillation. No significant findings noted, other than heart rate of 58. Patient has had no cardiac symptoms and mental condition has remained excellent after his ECT of April 1947.

revealed no abnormalities and an electrocardiogram showed no significant findings (Fig. 3).

COMMENT

The case presented is of particular interest inasmuch as the patient developed auricular fibrillation with electroconvulsive therapy after careful preliminary study had revealed no serious physical condition, and yet, following his second electroconvulsive treatment, he developed a severe disturbance of cardiac rhythm. It was believed that this was directly associated with the electroconvulsive therapy, and a series of electrocardiograms substantiated this.

Arrhythmias following metrazol and insulin shock therapies might be explained as due to toxic damage to the myocardium. Such damage results in changes in the refractory period which, according to Bellet, Freed, and Dyer (3), lead to fibrillation and which are believed by Hoffman (2), to be the cause of the fibrillation reported after metrazol therapy. The profound changes in the general circulation that accompany grand mal, together with the latter's violent muscular exertion, put a strain on the heart that can prove dangerous in the presence of any weakness, especially when coronary disease is present, according to Jetter (4).

Other explanations might be given for the cause of auricular fibrillation occurring as the result of excessive central nervous system stimulation. Korth (5) mentions that organic lesions (apoplexy, concussions, tumors of the central nervous system), and severe emotional upset may give rise to certain cardiac arrhythmias, such as extrasystoles and changes in auriculo-ventricular conduction.

SUMMARY

1. A case of transient auricular fibrillation in the use of electroconvulsive therapy is presented.

2. Electrocardiograms were taken prior to the electroconvulsive therapy and during the clinical manifestations of the fibrillation. An electrocardiogram was repeated 4 months after the auricular fibrillation occurred. Evidence of permanent cardiac involvement has been persistently negative.

3. A review of the literature has failed to reveal auricular fibrillation as a complication of electroconvulsive therapy.

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COMMENT

CONTROL OF JUVENILE DELINQUENCY

To consider and recommend a program of specific action in the various fields that touch on juvenile delinquency problems, a conference was called by the Attorney General of the United States in November, 1946. This was the National Conference on Prevention and Control of Juvenile Delinquency, attended by over 800 active participants. These included representatives of Federal, state, and local governmental agencies and private welfare groups that are national either in scope or in influence. Also invited were certain individuals not affiliated with particular organizations, who were in a position to contribute to the program against juvenile crime, and, in addition, representatives of organized labor, farm groups, business associations, fraternal orders, church groups, and other nonprofessional public organizations.

The work of the conference was accomplished through individual panels meeting simultaneously and reporting their recommendations back to the conference. Final reports prepared by 15 panels were presented to the plenary session and approved by the conference after full discussion. These reports have been separately published by the U. S. Government Printing Office and are obtainable from the Superintendent of Documents, Washington 25, D. C. They have been summarized in a pamphlet entitled, "The National Conference on Prevention and Control of Juvenile Delinquency: Summaries of Recommendations for Action." Each of these reports is a "tool for action," primarily at the community level. They state the problems in the particular field covered and illustrate, to the fullest extent possible, how the problems have or have not been met. General and specific recommendations are made for action by the members of the community.

The recommendations of the different panels will repay careful study by those interested in the various aspects of the control of juvenile delinquency. Subjects covered include the following in all their ramifications: community coordination; institutional treatment of delinquent juveniles; juvenile court laws, administration, and detention facilities; the rôle of the police in juvenile delinquency; recreation; housing, community development, and juvenile delinquency; youth and citizen participation; mental-health and child-guidance clinics; case-work and group-work services; responsibilities of church, school, and home; statistics; and rural aspects.

The National Conference created a continuing committee on the prevention and control of juvenile delinquency. This committee is developing and executing plans and programs to carry out the recommendations of the National Conference at Federal, state, and local levels. It collaborates with private agencies and with groups, governmental and nongovernmental, having responsibilities or facilities relating to juvenile problems, and makes available information regarding material and resources that relate to the recommendations of the National Conference.

On January 27, 1948, President Truman issued a proclamation designating the month of April, 1948, for the holding of state and community conferences on juvenile delinquency. In keeping with this Presidential Proclamation, the National Conference offered its help to the governors of all states and territories and the mayors of more than 1,000 cities, in order that such conferences and local projects might be organized; response was decidedly encouraging. A "Handbook on State and Local Conferences," which describes the first steps in organization, is being distributed by various national groups to their local membership. Up to 1,000 copies of the handbook may be obtained free of charge by writing to Eunice M. Kennedy, Executive Secretary, National Conference on Prevention and Control of Juvenile Delinquency, Office of the Attorney General, Washington, D. C.

A concerted attack on the problem of juve-

nile delinquency is now at hand. Though the major work must be done at community levels, those who devote time and energy to this task owe much to the guiding principles that came out of the National Conference. Its continuing committee, composed of more than 50 individuals, offers invaluable professional opinion, as well as information, which will help to coordinate the efforts of the nation to meet the needs of youth.

NOTICE

Attention of correspondents is called to the change of address of the editorial office from 111 St. George St., Toronto, to 113 St. Clair Ave., West, Toronto 5, Canada.

NEWS AND NOTES

THE 1948 LASKER AWARD.—Dr. George S. Stevenson, medical director of the National Committee for Mental Hygiene, has announced that the 1948 Lasker Award of $1,000 for outstanding service in mental hygiene will be presented at the annual meeting of the National Committee to be held on November 3 and 4 in New York City. The award will be presented for a recent significant contribution to the education of the physician in the psychological aspects of the practice of medicine (by "physician" is specified the nonpsychiatric medical practitioner). The work of the candidates for the award must have been accomplished or generally accepted during the past year or two.

Anyone may submit nominations, which should be forwarded by September 1 to the National Committee for Mental Hygiene, at 1790 Broadway, New York 19. Further information will be supplied on request.

DRAMATICS PROGRAM AT VA HOSPITAL.—Patients with mental illnesses in the Veterans Administration hospital at Coatesville, Pa., are taking part in a program of dramatics which, in addition to acting, includes training in property making, costume designing, scenery designing, casting, directing, and script writing. Several one-act and three-act comedies and dramas and a musical review have been presented in the hospital auditorium; and small units within the theater group have given a number of skits for the entertainment of fellow-patients unable to leave their wards.

The dramatics program is under the direction of Mrs. Carolin Witherspoon, a volunteer worker associated with the American Theater Wing, who during the war organized similar programs in army hospitals.

NATIONAL INSTITUTE OF PSYCHODRAMA.—A Conference on Action Methods and Training in Human Relations, sponsored by the Moreno Clinic and the Sociometric Institute, will take place at the Therapeutic Theatre in Beacon, N. Y., May 28-30, 1948. Subjects will be psychodrama (first day), sociodrama (second day), and sociometry and group psychotherapy (third day).

Summer seminars of the National Institute for the training of students in the above subjects begin June 1 and last until October 31. For further information, write to the Moreno Clinic, Beacon, N. Y.

NATIONAL HEALTH ASSEMBLY.—This Assembly, which is meeting in Washington, D.C., May 1 to 4, has been called for the purpose of formulating and reporting to the President a national health program for the next 10 years. Professional and lay leaders concerned with all aspects of health will advise the Federal Security Administrator, who is convening the Assembly.

The work of the Assembly will be carried on by 14 sections dealing with the major health problems of the nation. These will include the subjects of professional personnel, hospital facilities, local health units, chronic disease, maternal and child health, rural health, research, medical care, community planning, rehabilitation, dental health, accidents, nutrition, and mental health. Dr. William C. Menninger will be chairman of the latter section, which will be composed of about 40 professional and lay members.

CARNEGIE CORPORATION GRANT.—The following grant was made recently by the Carnegie Corporation: $30,000, payable over 3 years, to the University of Chicago for a study of psychological and sociological problems of later maturity. The grant will enable the University to continue exploratory studies in this field, under the direction of Professors Ernest W. Burgess, Robert J. Havighurst, and Herbert Goldhamer.

Further information may be secured from Professor Robert J. Havighurst, Committee on Human Development, University of Chicago, Chicago 37, Illinois.

BOOK REVIEWS

FROM CALIGARI TO HITLER, A Psychological History of the German Film. By *Siegfried Kracauer.* (Princeton University Press, 1947.)

Dr. Kracauer has made a study of the important productions of the German cinema between the two wars with the view of revealing the psychological forces operating in Germany which set the stage for Hitlerism. It is his thesis that films are a more direct representation of a nation's mentality than other modes of artistic expression because they are the products of many individuals and they must satisfy the multitude. This is a thoroughgoing and fascinating piece of research. The insight which it gives might be put to use in the treatment of postwar Germany.

Just after the end of the first World War there was a rash of pornography and films which resembled a "prisoner's day dreams." Psychologically the German people had withdrawn themselves from the outside world. In 1920, there appeared the archetype of the postwar productions, "The Cabinet of Doctor Caligari." This horrifying story of the madman who sent his hypnotized victim about on missions of murder was a foreboding of things to come. As in the Nazi world, there was a sinister atmosphere of terror in the film. Significantly, in the original form Doctor Caligari represented insane authority which was ultimately exposed and overthrown, but on the screen he was a benevolent psychiatrist. His crimes were nothing more than the delusions of one of his patients whom he promises to cure. In this and other films, the author finds evidence that the German soul was wavering between tyranny and chaos. "The Golem" and "Homunculus" were loathesome monsters who repeated the tyrant theme. Across the screen went a parade of fantastic crimes and tortures which later came to life in Nazi Germany.

The psychiatrist will probably find this book tedious at times, unless he has a particular interest in the technical side of the film industry. There is more history than psychology in the work, and the interpretations are for the most part on the sociological level. It is a volume packed with carefully documented material. There are many fine illustrations and copious footnotes.

A reprint of the author's pamphlet, "Propaganda and the Nazi War Film," which was issued in 1942, is added as a supplement. In it he gives a detailed analysis of the Nazi technique of using the film as propaganda.

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A TEXTBOOK FOR PSYCHIATRIC ATTENDANTS. By *Mrs. Laura W. Fitzsimmons.* (New York: Macmillan Co., Ltd., 1947.)

Mrs. Fitzsimmons has provided a manual particularly valuable for the large group of individuals who have not had regular nurses' training courses—the psychiatric aides and attendants. It is a ready reference for such subjects as special psychiatric problems, treatments and procedure, emergencies, ward housekeeping, and special problems associated with mental patients.

The book should be a useful addition to every psychiatric hospital library.

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WAR STRESS AND NEUROTIC ILLNESS. By *Abraham Kardiner,* with collaboration of *Herbert Spiegel.* (New York: Paul B. Hoeber, Inc., 1947.)

The first edition of this book published in 1941, based on the senior author's long and intensive experience with traumatic neuroses, was prepared as he puts it in anticipation of problems expectable in World War II. The new edition (428 pp.), done with the collaboration of Dr. Spiegel, brings the presentation up to date on the basis of the actual experience of this war and intensive work with cases incident to it. The approach is very thoroughgoing and comprehensive and at the same time clear, practical, and informative, with abundant helpful case illustrations. The chapters orienting the war situation as a social strain, discussing the genesis of neurotic reactions through it, the nature and treatment of the acute syndromes encountered and of after-following chronic states, also those sections dealing with psychodynamics and the development of the "effective ego" are especially noteworthy. Of value, too, are the discussions respecting the use of barbiturates and hypnosis as means of entree to critical aspects, in addition to what may be gained for the progress of the case through the direct conscious approach. In the opinion of the reviewer this work is a definite and timely contribution and is recommended for careful reading to those interested in the general question of neuropsychiatric difficulties traumatically induced and, particularly, disturbances of this order likely in present-day war or equivalent acute social conflict situations.

T. R.

THE PSYCHOLOGY OF EGO-INVOLVEMENTS. By *M. Sherif and H. Cantril.* (New York: Wiley, 1947.)

Sherif and Cantril are among the foremost social psychologists of the present day. They have come to the problems of ego-psychology from the vantage point of authoritative work in the measurement of attitudes, experiments on the formation of social norms, and other studies of social behavior. Consequently, one cannot expect that they would view the structure of the personality in the same way as the clinician who approaches the same theoretical problems via his intensive work with indi-

vidual persons and their difficulties. Usually in such a situation, each of the differently oriented parties has so much "ego-involvement" with his own approach that he can see little merit in the other, and mutual hostility may develop.

Unfortunately, the present volume is no exception. The authors have a good deal to contribute to a unified ego-psychology, which must account for the conscious, everyday behavior with which the book is mostly concerned, but they find it difficult to assimilate anything from the other main stream of ego-psychology, psychoanalysis. They examine a number of excursions into social psychology made by psychoanalysts, and conclude from the inadequacy of these analysts as amateur social psychologists that psychoanalytic theory must be completely rejected. Just so, the dynamically oriented reader may be tempted to draw similar conclusions about Sherif and Cantril's theories of the individual ego, its development and breakdowns. In both cases, the overlooked difficulty in understanding is the lack of an area of shared experience in which workers with different orientations may pool their varied contributions. Such a synthesis is needed and will come in time; *The Psychology of Ego-Involvements* fails to achieve it.

The book essentially starts from the social psychological observation that much of people's everyday behavior is importantly determined by strong values and identifications. When the patriot's flag is insulted, he feels a sense of personal outrage; when the alumnus' team wins, he gets a sense of greater self-esteem and importance. Phenomena of this kind, Sherif and Cantril argue, are *par excellence* ego-experiences, and the personal values that cause such intense reactions to events must be constituents of the ego. So far, it is easy to follow them. But they conclude that the ego is nothing more nor less than the aggregate of all such values and attitudes. What then happens to all the perceptual, defensive, and integrative functions that have usually been called *ego* by dynamic psychology and psychiatry? The authors have a good deal to say about perception—in fact, one of the book's most solid contributions is its emphasis on the perceptual stage in attitude-formation—but their conception of the ego is consistently structural, not functional and consequently not dynamic. They promise to take up in the future the problem of motivation (and also, one may hope, the rest of ego-psychology); perhaps they will be able to produce a fresher and more useful conceptualization than the ones they reject. That they will seems doubtful, judging from the nature of their criticism of psychiatric theories, and other indications of their methodological position, which seems to be a confused one. For example, early in the book they declare that it is necessary "to develop concepts which have scientific generality of the kind found in the natural sciences" and "functional variables." Later on, however, not only do they fail to develop such concepts, but one of their main criticisms of psychoanalysis is that its concepts are "too abstract"—that is, have scientific generality.

On the positive side, the book contains a long and fairly comprehensive review of today's attitude psychology, a complete survey of experimental studies of ego-involved behavior, and a summary of many important sociological studies of groups and their effects on individual members. The authors seem, however, to have sacrificed thoroughness in understanding and accuracy for exceptionally broad coverage; in at least two cases, a position is attributed to an author which he has mentioned only to refute it, and the level of understanding of psychoanalytic theory may be judged from the denial that unconscious motivation is socially important since in "newspaper accounts of industrial strife . . . . both labor and management seem very definitely to know what they want." A novel feature of the book is a chapter of "illustrations from literature," in which selections from a variety of fictional works, ranging from *Antigone* to *Kitty Foyle* are interpreted in accordance with the authors' theories.

For the above reasons, and because the kind of phenomena with which it is basically concerned are of importance, the book might be recommended to the psychiatric reader with some reservations. He will be introduced to many empirical studies he might not otherwise come across, but he will not find a clarification of ego-psychological problems in the theoretical chapters. He may find the others easier reading if he substitutes, in most of the places where the authors use the word *ego*, E. H. Erikson's happy coinage, *ego-identity*—a concept which seems to deal with the same phenomena in a more useful and fruitful way.

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PERSONALITY AND ITS DEVIATIONS. By *George Herbert Stevenson, M.D., F.R.S.C.*, and *Leola Ellen Neal, M.A., Ph.D.* (Springfield, Ill.: Charles C. Thomas, 1947.)

This book is interestingly compiled in that, when there is a change of subject matter, reference is made to the preceding chapters and their contents generalized. There is then a generalized statement regarding what may be anticipated in the present and succeeding chapters.

There is much more emphasis than usual on the physical factors which may enter into the development of one's personality, and these are dealt with in a clear, factual way with excellent illustrative material to clarify further the points to be made.

Phases of personality development and functioning about which there are controversial ideas are discussed in a broad general way, and then the ideas of the authors are clearly defined.

Hereditary factors, both physical and mental, are given a strong place in the personality potentials, and then the influence of environmental factors on these potentialities is discussed in much detail.

From beginning to end, one's interest is held with increasing tenacity; and, although primarily written for lay and nonprofessional use, because of the rather exhaustive reference list for related reading

at the end of each chapter this book should make a highly desirable addition to any psychiatric library.

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NURSING IN MODERN SOCIETY. By *Mary Ella Chayer, R.N., M.A.* (New York: G. P. Putnam's Sons, 1947.)

The shortage of nursing services is currently of great interest to the general public. It is sometimes implied that nursing organizations are at fault or that nurses themselves are devoid of a sense of responsibility. Before we can hope to establish conditions satisfactory to both the nursing profession and the public, we must first understand the problem. Miss Chayer has prepared a valuable contribution toward that understanding in her book, "Nursing in Modern Society." It is timely, well organized, and objective, and should prove helpful to harassed hospital administrators, directors of public health organizations, and others utilizing nursing services. The general public, too, especially young people considering nursing as a career, will find its well-presented facts rewarding.

The book is constructed in three parts. The first part is concerned with the impact of social forces on nursing and with an analysis of nursing as a social force. The background of the problem and some factors which influence it are presented. The second part deals with a study of the influence of social forces upon community health needs; and the third part, with what Miss Chayer calls "Building a Better Future." In the last section she presents the objectives of the World Health Organization, while discussing the responsibility of world citizenship for nurses as well as others.

Miss Chayer points out the challenge to the International Nursing Council in playing a more effective role in international affairs. This organization was established 50 years ago and has 40 member nations. It has weathered two world wars and is struggling back into activity. Miss Chayer believes that, with wider knowledge of its existence and a more vigorous program of activity, the International Nursing Council can become a potent constructive force on an international scale.

Miss Chayer presents viewpoints that may revive old controversies. It is suggested that nursing education be established on levels, and that students progress from the lower to higher in succession, as pupils progress from kindergarten to elementary to secondary to university educational levels. When nursing was young in America, it was the practice in some mental hospitals to employ attendants and give them some lectures in a variety of subjects. Those who passed examinations were graduated as nurses and employed as such. In some cases the results were satisfactory, and professional nursing benefitted by the acquisition of a valiant, effective leader. In other cases the result was less desirable. Some mental hospitals continue their basic schools and some continue to recruit nurses from the attendants. The divergence between good and poor results continues to be great.

The text ends with a statement concerning nursing in the social structure of today, stressing the need for every nurse to acquire an awareness of the changes taking place in the special organizations serving humanity and the "common goals toward which education, health, and social welfare must bend their efforts if life's deeper purposes are in some measure to be fulfilled."

Questions for study and suggestions for further reading follow the chapters. An extensive bibliography is included at the end of the text.

"Nursing in Modern Society" is the first of a series of texts on modern nursing, edited by Genevieve Knight Bixler and published by G. P. Putnam's Sons. Miss Chayer's book sets a standard of excellence which will challenge future writers in the field. If the texts that are to follow will be of as high caliber as this, one can anticipate a notable contribution to nursing literature.

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PSYCHOTHERAPY IN CHILD GUIDANCE. By *Gordon Hamilton.* (New York: Columbia University Press, 1947.)

This book, by a professor of social case work in the New York School of Social Work, suffers from a diversity of purpose. Among its various aims are to answer the questions, "In what way is psychotherapy related to social case work, and may it be practiced in a social work rather than a medical setting, and, if so, with what safeguards and controls?"; to describe adaptations of therapy carried on by social workers at the Jewish Board of Guardians (New York City); and to add to the few authoritative texts on psychotherapy with children. These diverse objectives are presumably reconciled by the fact that for many years at the Jewish Board of Guardians social workers have engaged in psychotherapy, with psychoanalysts acting as teachers, consultants, and participants in diagnosis. A description of their work and of the principles underlying it, set forth in textbook fashion, constitutes the major portion of the volume. From this and from the fact that many clients of social agencies are emotionally maladjusted it is concluded that psychotherapy should be added to the already numerous types of activities of social case work.

It is beyond this reviewer's competence to comment extensively on the psychiatric principles and methods described. Many interesting and suggestive statements are made, and the diagnostic categories, on which much emphasis in treatment is placed, are convincingly described. It seems, however, that many statements are made too dogmatically and that the Freudian basis of the whole work is not made sufficiently explicit. This, of course, is a criticism of the book as a text; as a criticism of what the Jewish Board of Guardians does and believes it would not be valid.

Regarding the practice of psychotherapy by social case workers there will be much difference of opinion among both psychiatrists and social workers. The question cannot be dodged by saying that

psychotherapy is a term broad enough to embrace the activities of several professions, for the work described in this book is clearly similar to the brief-treatment methods that some child analysts employ and there is little limitation to the types of disorders worked with. It must be noted, however, that this treatment is carried on in an organization set up to serve problem children (its clients therefore come to it for that service) and that the social workers are specially trained and work under psychiatric direction. If results comparable to those of psychiatrists are secured (a question which this book does not attempt to answer) a way out of the current shortage of child psychiatrists may have been found.

The question at issue, in this reviewer's opinion, however, is whether psychotherapy should be carried on under the aegis of social work, as one of its accepted professional activities. There are various reasons for thinking the answer should be in the negative. The one that psychiatrists may be most interested in is that which concerns licensing or other forms of control. While the work of the organization described in this book is adequately safeguarded and the author's proposals also contemplate psychiatric control, there is really no reason why workers so trained should not be engaged by ill-equipped social agencies and, being actually only psychiatric technicians rather than fully educated therapists, do considerable damage in some cases. What is needed, it seems to me, if this work is to be enlarged is the development of a new professional category with a standardized and regulated relationship to that of psychiatry. For admission to training for this new profession social case work would undoubtedly be a good preparation, as might clinical psychology, child development, and progressive education. The special knowledge and skills which such students would possess would enrich this new effort and would perhaps benefit psychiatry itself.

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Laboratory Manual of Microbiology for Nurses. By *Elizabeth S. Gill* and *James T. Culbertson.* (New York: G. P. Putnam's Sons, 1947.)

This manual presents bacteriological laboratory methods fundamental to the understanding and appreciation of microorganisms commonly pathogenic in man, means of diagnosis, modes of transmission, isolation technique, and surgical asepsis. The material is pertinent, concise, and complete as to subject matter and procedure. It is conveniently arranged for beginning students so that it could be completed in 16 to 18 two-hour laboratory periods.

Especially desirable features are:

1. The construction of the notebook from which pages can easily be removed for grading and to which they may be returned for future reference. This is time-saving for both student and instructor during the busy preclinical period.

2. The questions at the end of each exercise which augment the student's knowledge and interpretation of the bacteriological facts and data.

3. The use of articles of ward equipment in the laboratory procedures on disinfection and sterilization, to couple them in the nurses' minds with the requirements for effectively treating each type of equipment in such a way that will, in the ward situation, recall to her the importance of fulfilling those requirements meticulously.

4. The inclusion of directions for making basic types of media commonly employed for the pathogens studied, and the lists giving sources of materials needed.

Advocation of the use of the Arnold sterilizer for sterilization of media and the necessity of clarifying extract and infusion agar with egg white will be questioned by many. The method of subculturing contaminated objects subjected to chemical disinfectants by streaking directly on an agar plate might lead to false conclusions concerning absence of growth. Bacteriostatic effect might prevent growth on an agar plate, when greater dilution of the disinfectant by another means of culture would show growth.

With the expanding knowledge of microbiology and its practical application to curative and preventive medicine goes a widening demand that nurses be well informed and competent in this field. The reviewer believes this laboratory manual will aid both student and instructor in achieving this goal.

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A Field Guide to The Shells (of our Atlantic Coast). By *Percy A. Morris.* (Boston: Houghton Mifflin Company, 1947.)

Anyone who is interested in the collection and study of sea shells as a form of occupational therapy will find this book useful. Among many handbooks available on the market today, this one offers some of the most unique advantages for practical use. It can be carried in the pocket. It is very complete and the illustrations are "out of this world." As a science hobby, conchology is coming more and more into its own. It is more widely recognized as a fascinating pursuit for anyone who may have the leisure and interest to give it. This new book by Professor Morris of the Peabody Museum of Natural History at Yale University will amply reward anyone who can spend some vacation time on a beach where shells abound, whether it be in Florida in the winter or Cape Cod in the summer.

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